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TREATMENT OF DETACHMENT OF THE RETINA

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The surgical treatment of detachment of the retina is undoubtedly the most interesting subject in ophthalmology today. It is to Gonin, the celebrated professor in Lausanne, that ophthalmologists owe the discovery of this treatment. He studied the condition for many years, and eventually his efforts were crowned with success. The importance of Gonin's discovery is enormous. The fact that about 20 per cent of the adult blind lose their vision through detachment of the retina, that this number was formerly practically incurable and that now through operation the trouble can be cured in the great majority of cases surely confirms the importance of the new treatment. I cannot speak of this subject without offering homage to this investigator, who, unfortunately, died two years ago.

Gonin was learned, sincere and good. Learned because he arrived at his discoveries not by hazard but as the result of prolonged study and many investigations. Sincere because he never wrote anything but the truth and wrote without the slightest exaggeration. This is possibly the reason why at the time of his first publications no great attention was given to them. The 20 to 25 per cent of favorable results which he then modestly noted have been proclaimed by other authors with other methods. Finally, Gonin was good. All those who knew the man intimately knew how he lived for his family, his friends and his patients. A last example was the bestowing of a great part of his modest fortune on the blind of Lausanne.

The subject is large, but so much of it is now well understood that I can limit myself to a brief article. I have found in traveling and in reading foreign articles that the subject is still difficult for many and frequently not well understood. To speak truthfully, the treatment of detachment of the retina has not been adopted as well as it should be. To my mind, the principal reason is that, owing to the diversity of clinical types, the operator must adopt operative measures according to the condition in each case. The majority of ophthalmologists perform a good operation for cataract or glaucoma, in which the procedure is gen-

erally the same in all cases, but in operations requiring various technics, such as dacryostectomy or operation for detachment of the retina, conditions are more difficult, and in atypical cases the operator is handicapped by lack of experience. As operation is really easy in the cases in which the condition is uncomplicated, it is necessary to begin by operating in this type of case and not hesitate, because success will encourage the confrères to operate with greater success in the more difficult cases. It is preferable, from a humanitarian point of view, that the majority of ophthalmologists operate with success in 50 per cent of the cases rather than that certain ones obtain good results in 70 per cent. The first group passes with time into the second.

The greatest difficulty lies in the ophthalmoscopic examination, for the procedure of operation depends on careful examination of the eyegrounds and on the ophthalmoscopic control during operation. When it is difficult to examine the eyeground on account of opacities, the prognosis is poorer, as the study of the eyeground and the retina is interfered with. Many of the colleagues are content with the results obtained by the relatively simple procedure (extensive operation in the suspected region, particularly large barrages). They obtain with this technic cures in 40 to 50 per cent of the cases, which, when compared with the results obtained by subconjunctival injections of sodium chloride, is, of course, marvelous. But if the colleagues will attempt to make a more careful localization of the area to be operated on, they will be able to improve their results in from 20 to 25 per cent of the cases.

Of course, a large barrage and an extensive operation are frequently indispensable, but it is important to try to do the operation with the least danger to the eye, as the intolerance on the part of the eye increases with the extent of the operation, and, moreover, it is desirable to sacrifice the smallest possible area of retinal territory. While it is not always possible to localize the lesion carefully, an attempt must be made with all the skill at one's disposal to obtain an accurate localization. This requires a prolonged and often fatiguing ophthalmoscopic examination. It is much easier not to wear oneself out in the dark room and do a large and not necessarily limited operation, but this is not so good for the patient.

OPHTHALMOSCOPIC EXAMINATION

Ophthalmoscopic examination should be made with direct and indirect images, with maximal dilatation of the pupil produced by instillation of atropine sulfate or epinephrine hydrochloride or by a subconjunctival injection of epinephrine hydrochloride. The indirect image requires a very intense light. The best source is the projection

lamp with the use of from 100 to 200 volts. In this lamp the filaments are zigzag and placed in the same plane. The glass may be either dull or clear. The indirect image is particularly necessary in examination of the extreme periphery of the retina. For the direct image most of the electric ophthalmoscopes are good. They must carry a correction up to 30 D. and have a good intensity of light. The direct image is indispensable for judging differences in level and in the definition of small details. The result of the ophthalmoscopic examination must be entered on a schematic drawing designed according to that of Amsler and Dubois. There is no other method of control for a good study of the case or in order to acquire experience. I have occasionally been called to operate on a patient with a good history, the description of whose symptoms and pathologic observations took up from eight to ten typewritten pages, but in whose case there had been no attempt to make a drawing. In other words, the most important thing remained to be done. I rapidly read the text, principally out of politeness, and then set about making a drawing. The necessity of a good drawing is important not only to serve as a guide during the operation but for postoperative control and to enable one to judge the results in general when the different cases are studied in groups according to the clinical character of the condition as shown by the drawing. A good drawing helps to determine the proper method of treatment and operation and is also an aid to prognosis and in acquiring experience. Ophthalmoscopic control makes the operation more effective and perfects one in the technic, as it is a help always to see what one is doing. Sometimes the postoperative results are surprising. One may expect a good result, and it does not take place. In other cases, a condition may seem difficult to cure, and the result of operation proves to be good. These surprises would be less startling if one had made a careful ophthalmoscopic examination during and immediately after the operation for the purpose of control.

When one sees that the electrodes have not touched the retina and, after the subretinal liquid has been allowed to escape, that the retina still remains far from the choroid and that folds in the retina are still present, the prognosis is not favorable.

In order to show the importance of ophthalmoscopic examination I shall report two unusual cases. In one of these the patient was a woman presenting a macular hole. While I was making a diathermic surface coagulation I coagulated at the same time the blood in the superior retinal vein, and on examining the eyeground was startled to see a thrombosis. The vision was lost in a sector of the field as the result of this vascular lesion.

In another case, in which there was a tear in the upper part of the eyeground, with bulging of the retina which had not become reduced

by rest in bed, an attempt was made with the long diathermic needle to grasp the retina and draw it toward the choroid. I then found with the ophthalmoscope that I had torn the retina from the place where the needle had touched it as far as the posterior pole near the optic nerve. Without ophthalmoscopic examination I should not have been able to understand the reason for my ill success.

REPAIR OF HOLES

The meridian is noted by means of a mark made at the limbus with china ink or with an alcoholic solution of gentian violet by a fine needle. The distance of the hole from the limbus is more difficult to determine. One may estimate the distance in papillary diameters, as has been done for a long time, but it is much better to orientate oneself with the ophthalmoscope after having made a galvanopuncture, or, better, a diathermic puncture or after having introduced a needle or a fine instrument into the eye, or with transillumination or diaphanoscopy. The old method of estimating the distance in papillary diameters between the ora serrata and the tear is not entirely exact, because the ora serrata is not always visible, and it is difficult to estimate the exact location of the ophthalmoscopic peripheral field. In most cases the distance in millimeters is measured with a compass on the sclera, and this is generally sufficient.

Thermopuncture with a fine galvanocautery may give good orientation, but it has the inconvenience of sometimes making a hole in the retina, the choroid and the sclera, which drains the subretinal cavity excessively.

Diathermic punctures are of great importance when the retina is not too bulging. The diathermic needle is introduced into the suspected region, and if one does not recognize with the ophthalmoscope any change in the retina in the form of a white patch some of the subretinal fluid is allowed to escape with the aid of pressure exerted on the eyeball. The needle is again introduced at the marked area, and ophthalmoscopic examination is repeated. Then in nearly all cases the white area will be discovered, and its relation to the tear can be determined.

If the retina shows bulging and this does not improve after the escape of the subretinal liquid so that the diathermic puncture is not visible, a small needle or knife may be introduced, which is more easily visible with the ophthalmoscope. Transillumination consists in throwing light on the tear with the ophthalmoscope. The assistant notes which part of the sclera is illuminated. When the operator sees that the tear is well illuminated, the assistant makes an electric puncture at this part of the sclera or applies china ink or gentian violet. In diaphanoscopy, on the other hand, a transilluminator, such as the lamp of Lange or

that of Rochon and Duvigneaud, is used. The sclera is illuminated while the operator is examining the eyeground, and when he perceives the light passing through the suspected area the assistant is asked to make a mark, or to pass the current, if the lighting apparatus carries the diathermic electrode as suggested by Strampelli.

Whenever the condition is simple, any of these procedures is good; but if the retina is very bulging and the vitreous is cloudy, the difficulties increase, and the operation is accordingly more difficult. In any case, all efforts to obtain good localization will be recompensed by better results of treatment.

ANESTHESIA

A local anesthetic is sufficient in most cases, and it is well to administer hypnotics. Instillations of an anesthetic should not be too frequent, in order not to change the corneal surface. The eye, of course, must be closed between instillations. The injection should be made into Tenon's capsule and not subconjunctivally.

OPERATION

Cautery.—The method of Gonin is useful in cases of very simple conditions in which it is possible to make an accurate localization so that the diathermic and perforating applications will fall just in the place of the tear. This method has been practically abandoned today on account of the danger of hemorrhage, which sometimes complicates the postoperative course.

Caustics.—The use of caustics is efficacious but very difficult, as it is hard to trephine the sclera without perforating the choroid. It is much more painful than other procedures, and one obtains as good results with other methods, especially diathermy. This procedure was suggested by Lindner and Guist.

Diathermy.—Diathermy is the most extensively used method at the present time. First designed by Weve, it was rapidly adopted by most operators on account of its freedom from danger. Diathermy may be applied in two ways—to the surface or perforating the surface. To apply surface coagulation one must use an instrument with a flat or a round tip, and the sclera then acquires a slightly grayish color. To obtain this result a current of from 30 to 60 milliamperes is necessary. Perforating diathermy requires a current of greater intensity—from 80 to 150 milliamperes, according to the condition and the size of the electrode. Each method has its definite indications. In general, one uses both methods. When the detachment is flat and the retina is not far from the choroid, surface diathermy followed by a small perforation to drain the subretinal fluid is the best method. In cases of disinsertion

near the ora serrata this method is usually satisfactory. In cases of macular holes it is prudent to use surface diathermy in the macular region very cautiously so as not to injure the retina.

In cases in which there is a large, bulging detachment, surface diathermy may be used associated with perforating diathermy to obtain drainage of the subretinal spaces. This drainage must be as much as the retroretinal cavity requires. An excessive bulging of the retina is an inconvenience; an attempt is made to reduce it before the operation by rest in bed and during the operation by compression of the eyeball after puncture of the sclera. In using perforating electrodes one may add a kind of surface diathermy without actually perforating the sclera. This is suitable if it is difficult to obtain dry surface areas, because for surface diathermy to be effective the sclera must not be moist.

In making diathermic punctures one should not make these too close to one another, as scars will result. It is always best to leave a space of 0.75 mm. between each puncture. This, of course, applies only to perforating diathermy. In surface coagulation the punctures may be made closer together. Vorticose veins should also be carefully avoided, experience having shown that their obstruction by coagulation retards healing after operation, notwithstanding the fact that the vessels in the choroid present many anastomoses.

The continuous barrages, when applied transversely, that is, parallel to the equator of the eye, have the inconvenience of coagulating the vessels in an ocular sector and obstructing the circulation for some time; they possibly also interfere with the function of the ciliary nerves. This is probably the reason why in cases in which the coagulation has been extensive there is difficulty in dilating the pupil. It is important that the drainage of the subretinal pocket exist long enough, because if the perforations close too early, before the liquid has been absorbed, the retina will not come into contact with the choroid, and adhesion will not take place. To insure satisfactory drainage the openings must be sufficiently large. The fine needles used in diathermy often close too rapidly and are applicable in cases in which the retina is only slightly detached. If there is considerable fluid the perforations should be made with a needle 0.5 mm. in thickness, or, when a fine needle is used, the perforating action should be prolonged and the borders of the hole enlarged. Galvanocautery may also be used to make a perforation for drainage or a trephine without endangering the choroid. The latter structure is then perforated with a blunt instrument, such as a strabismus hook. It is very necessary (and in this lies the secret of success) that the retina should be in contact with the choroid at the end of the operation. Nothing will produce adhesive choroiditis if the two membranes remain separated by a layer of subretinal liquid instead of being in

contact or slightly separated by an exudate produced at operation. If the galvanopuncture is going to be used for drainage, it is well to make a circle of diathermic applications first so that the vessels are coagulated. If the diathermic punctures are made where the surface coagulation has been previously done the sclera will be found much thickened. Personal experience, as well as seeing colleagues operate, helps one in judging the suitable degree of intensity for diathermy. The milliamperimetric measures are, of course, only relative. It is better to judge the effect by the change produced in the tissues, particularly with the ophthalmoscope. For surface coagulation, pyrometric electrodes are excellent. In cases of inferior or lateral detachment one may facilitate the adaptation of the retina by making an injection of air at the superior pole.

POSTOPERATIVE TREATMENT

When the operation is finished, atropine sulfate is instilled and a binocular bandage applied. The patient must remain in bed for several days, and it is always well, as Gonin has suggested, to incline the head of the patient to the side on which the operation has been performed. If the operation was done in the lower half of the retina the patient may be allowed to sit up partly. The length of time in bed depends on the condition. In general, from six to eight days are sufficient; in case of severe involvement this time may be lengthened, though it is more important to keep the eyes quiet than the body, because it is the movement of the eyes which causes trouble for the retina. It is best not to uncover the eye which has not been operated on too soon, as movement of the eyes will interfere with the healing of the retina. Stenopeic spectacles may limit the movements of the eyes, but the binocular bandage is much more efficacious. The dressing should be changed every three days and atropine sulfate instilled. On the sixth day the eyeground may be examined, and if progress is favorable and the condition is not severe the patient may be given some liberty. It is usually not wise to remove the binocular bandage before twelve days. If it is necessary to repeat the operation it can be done after this time. In general, I permit my patients to leave off their bandage and to wear stenopeic glasses after twelve to twenty days, according to the condition.

PROGNOSIS

The prognosis of detachment of the retina depends on the clinical variety of the condition in each case. In simple cases in which there are small holes, without a great degree of detachment and in which the detachment is of only a few days' standing, recovery is the rule. In cases of detachment with a tear at the ora serrata the condition is always easy to cure, but the extent of the detachment, the number of holes, the

duration and, particularly, disturbance of the vitreous make the condition difficult to cure in proportion to the development of any of these factors.

Myopia does not play a large rôle, though in cases in which the retina is very atrophic there is a greater tendency to recurrence. The age and general state of health of the patient also are important. Finally, the process of absorption and cicatrization will depend on the health of the eye.

PATHOGENESIS

A new problem which the interest in the modern treatment of detachment of the retina has created is that of the pathogenesis. The rôle of the retinal holes in the development of a detachment is incontestable; but the difficulty is to interpret how these perforations act. A number of theories have been advanced, but they do not hold for all cases. Undoubtedly there are several causes for detachment of the retina. I refer not to the cases of detachment without holes following exudative choroiditis, but to the great majority of cases, in which there are holes. In these cases the pathogenesis or the process of formation of the holes may proceed in one of two ways: by adhesion of the retina to the vitreous and detachment on movements of the eyeball or by adhesion of the retina to the choroid and choroidal exudation; the latter is much less frequent than the former. Pathologic examination of eyes with recent detachment has shown that the retina is atrophic and is much altered in each case and that through succussion or movements of the eyeball the retina becomes detached. To my mind, the adhesions which exist between the framework of the vitreous and that of the retina are the agents which produce the tears. It is well known that inflammation of tissues, if it lasts for any length of time, leads to atrophy and fibrosis of these tissues and adhesions to the adjoining tissues. Anatomic examinations of eyes with recent detachment have shown atrophy of the retina in some cases and adhesions to the vitreous and particularly pigmentary inclusions in the retina in others, which demonstrates the preexistence of an inflammatory process in both membranes. The inflammatory process, however, must not be so intense that the retina becomes adherent to the vitreous and not to the choroid. In the latter case the retina would not become detached. This explains detachment in cases of old choroiditis in which the inflammation is mild (black areas in the eyeground) and its absence in cases in which there are white patches, for in the latter the inflammation has been intense and has even destroyed the choroid, so that the two membranes have become adherent. There is a simple experiment which will demonstrate the ease with which the vitreous will adhere to the retina when the latter membrane is inflamed. After removal of the conjunctiva in a rabbit and irritation of the sclera at the level of the equator with either chemical or physical agents, if

the eye is removed after several days or weeks and placed in solution of formaldehyde U. S. P. and then in alcohol, the vitreous retracts as a result of dehydration. One can then see that the vitreous is much retracted but remains adherent to the retina at the place of irritation. I think that the adhesion between the vitreous and the retina develops in this way, and then a succussion or movement of the eye detaches a piece of the retina. On movements of the eyeball, more and more vitreous passes behind the retina. There may be tears produced by choroidal exudation. There must be a previous adhesion between the retina and the choroid to produce a rupture of the retina by raising of the retina in the periphery. This is, however, a much rarer phenomenon than the former.

CONCLUSION

In conclusion I cannot do better than repeat what I said at the beginning of this article and express my gratitude and render homage to Gonin, that benefactor of mankind.

TREATMENT OF OCULAR TUBERCULOSIS

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The results of treatment with tuberculin in 42 cases of ocular tuberculosis in the Wilmer Ophthalmological Institute were analyzed and reported in 1928.[1] The statement was then made that the manner in which tuberculin acted, whether as a specific desensitizing agent or by augmenting the specific local resistance by a succession of small stimuli, was not known. Since this report our knowledge of the influence of allergy and immunity on tuberculous lesions has been considerably clarified. We have therefore reviewed the cases of all patients with ocular tuberculosis treated with tuberculin or by other procedures in the public wards or in private practice in the Wilmer Ophthalmological Institute and have analyzed these cases in the light of this newer knowledge of tuberculosis. The study is the subject of this report.

The influence of allergy and immunity on local tuberculous lesions has already been discussed by one of us [2] elsewhere and will be further elaborated in the reports of general studies now in progress. For our purposes here the subject may be summarized as follows:

INFLUENCE OF ALLERGY AND IMMUNITY ON TUBERCULOUS LESIONS

Tuberculous lesions may vary between the acute inflammatory caseating lesions seen in highly allergic animals, the slowly progressive lesions of the normal animal and the rapidly limited, quickly encapsulated lesions in the highly immune animal. This wide difference is seen also in the lesions of ocular tuberculosis. On the other hand, there are the acute caseating necrotizing lesions with a tendency to perforate the globe, and at the other extreme, the self-limiting lesions which

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1. Woods, A. C., and Rones, B.: The Therapeutic Use of Tuberculin in Ocular Tuberculosis, South. M. J. **21**:613, 1928.

2. Woods, A. C.: Allergy and Immunity in Ophthalmology, Baltimore, Johns Hopkins Press, 1933.

heal but have a tendency to recur. Experimental studies in both general and ocular tuberculosis have shown that with similar dosage and virulence of the tubercle bacilli this difference in the lesions is due to the underlying hypersensitivity and immunity of the infected animal.

Since the time of Koch it has been recognized that normal and tuberculous animals react differently to tubercle bacilli. Under parallel conditions, the normal animal reacts with the formation of slowly developing tuberculosis with tubercle formation, while the tuberculous animal shows an acute localized inflammatory reaction, inhibition of dissemination and some actual destruction of the invading bacilli. The acute inflammatory reaction has long and correctly been recognized as due to hypersensitivity of the tissues of the tuberculous animal. The tuberculous animal becomes allergic to tuberculoprotein. It was believed for many years that the inflammatory allergic reaction was responsible for the resistance to further infection shown by the tuberculous animal and that this reaction fixed the bacilli at the site of injection, prevented their dissemination and aided in their encapsulation and destruction. Under this conception, allergy was responsible for immunity. In 1929 Rich and McCordock [3] sharply challenged this view and showed there was no experimental evidence for its support. In a series of experiments they demonstrated that allergy and immunity were two distinct entities, in no way related. Allergy was responsible for the inflammatory, exudative and necrotizing phases of tuberculous lesions, while immunity was responsible for the fixation of the bacilli and their encapsulation. Extending their researches to other infections, they showed that the allergy could be entirely removed by desensitization of the experimental animals, while the immunity was undisturbed. In 1933 Rothschild, Friedenwald and Bernstein [4] illustrated the same fact for tuberculosis.

The nature of immunity is not entirely clear; certainly there are two factors—a humoral and a cellular factor. The humoral element is concerned with the immune antibodies which tend to fix and immobilize the bacilli, while the cellular element is concerned chiefly with the macrophages which engulf and destroy the bacilli. Other factors, the immobilizing effects of the fibrin barrier and possibly certain physiochemical factors in the inflammatory lesions, may be concerned in immunity. Therefore, in the light of this knowledge that allergy incites

3. Rich, A. R., and McCordock, H. A.: An Enquiry Concerning the Rôle of Allergy, Immunity and Other Factors of Importance in the Pathogenesis of Human Tuberculosis, Bull. Johns Hopkins Hosp. **44**:273, 1929. Rich, A. R.: The Demonstration that Allergic Inflammation Is Not Necessary for the Operation of Acquired Immunity, Proc. Nat. Acad. Sc. **16**:460, 1930.

4. Rothschild, H.; Friedenwald, J. S., and Bernstein, C.: The Relation of Allergy to Immunity in Tuberculosis, Bull. Johns Hopkins Hosp. **54**:232, 1934.

the spreading, destructive, inflammatory phases of the tuberculous reaction and immunity is responsible for the healing and encapsulation of the lesion, in the attack on a local tuberculous lesion one's primary duty is to remove the fatal hypersensitivity of tissue and promote immunity.

THERAPEUTIC ACTION OF TUBERCULIN

There are obviously two different conceptions of the mode of action of tuberculin on local tuberculous lesions. For want of better names we term these the "perifocal" and the "desensitization" concept. The perifocal concept is founded on the old view that allergy is responsible for immunity and assumes that tuberculin excites repeated minor perifocal reactions about the local lesion, these repeated reactions producing local immunity which promotes encapsulation of the lesion and destruction of the bacilli. On the basis of this perifocal concept, tuberculin should be used in doses just beneath the point of focal reaction. Inactivity of the lesion is interpreted as the attainment of a local immunity, and, no further perifocal reactions being desired, tuberculin therapy is abandoned. The desensitization concept is founded on the idea that allergy is responsible for the inflammatory phases of the tuberculous lesion and is unrelated to immunity. Through removal of the fatal tissue hypersensitivity, which is responsible for the inflammatory and destructive phases of the tuberculous lesion, the bacilli are reduced to the status of inert foreign bodies, and the immunity acquired by the tuberculous infection, unrelated to allergy, effects their encapsulation and destruction. On the basis of this concept, tuberculin is used in small doses, especially in the presence of an active inflammation, and is continued in increasing doses over a long period after the lesion is apparently healed, the fundamental idea being to achieve and to maintain desensitization of tissue and to prevent the return of hypersensitivity which invariably follows the premature cessation of the use of tuberculin.

It is obvious that tuberculin used on the basis of the perifocal concept of action will have a definite desensitizing effect; in fact, more rapid desensitization may possibly be accomplished by giving tuberculin in quantities sufficient to provoke even minimal reactions. Therefore, whether tuberculin is given with the idea of producing perifocal reactions or with that of desensitizing the ocular tissues, primary desensitization of the same degree will probably ensue. This is the probable explanation for the temporary good effects of tuberculin reported by so many authors. The salient differences between the use of tuberculin according to the perifocal concept and that according to the desensitization concept are, first, the radically different views underlying its use and, second, the fact that under the perifocal concept the use of tuberculin is aban-

doned when inactivity of the lesion is obtained, while under the desensitization concept tuberculin must be used for long periods, and the dose increased steadily during periods of remission when the eye is quiet, to maintain desensitization which otherwise almost invariably would recur.

MATERIAL

The cases which form the basis of this report are, first, the 42 cases reported by Woods and Rones in 1928, in 4 of which the patients have been followed consistently since that date and treated on the basis of the desensitization concept of the use of tuberculin, and, second, 111 patients seen in the public ward and dispensary and 22 patients from private practice for all of whom the final diagnosis was ocular tuberculosis and who were treated with tuberculin in the Wilmer Ophthalmological Institute since 1926.

DIAGNOSIS

The diagnostic problem of ocular tuberculosis will be fully discussed in a later communication. For these patients the diagnosis was reached on the basis of the usual criteria: the history, the clinical picture of the diseased eyes, the tuberculin reaction of the individual patients and a thorough physical examination to determine the possibility of systemic tuberculosis and to eliminate other etiologic factors such as syphilis and focal infection. A Wassermann test and special examinations of the teeth, tonsils, accessory nasal sinuses and genito-urinary system were done as a routine. Special bacteriologic examinations and sensitivity tests were done when indicated. All foci of infection, such as periapically infected teeth, infected tonsils or sinuses and infections of the genito-urinary tract, were eradicated as far as possible. The tentative diagnosis of ocular tuberculosis was made final only after the effect of removal of foci of infection had been observed and it was clear the removal of such foci had no effect on the ocular lesion and did not appear casually connected with it.

The most pertinent point in the diagnosis was the tuberculin reactions of these patients. The Mantoux or intracutaneous test with graded amounts of tuberculin, 0.001, 0.01 and 0.1 mg., was done as a routine. On account of the danger of focal reaction the subcutaneous and the Calmette test were never used, and the von Pirquet test was likewise not employed on account of its extreme sensitiveness. In addition to the 175 patients who form the basis of this report, there were 5 additional patients with ocular tuberculosis not treated with tuberculin and 10 patients with ocular tuberculosis proved by histologic examination of the enucleated eyes [5] for whom we have records of the tuberculin reac-

5. These are the 10 patients previously studied by Friedenwald and Dessoff.

tions. The results of the Mantoux test in these 180 clinical cases of ocular tuberculosis and the 10 cases of histologically proved tuberculosis are shown in table 1.

Of the 180 patients with clinical ocular tuberculosis, 96, or 53.4 per cent, reacted to 0.001 mg. of tuberculin; 75, or 41.6 per cent, had cutaneous reactions only to 0.01 mg., and 9, or 5 per cent, reacted only to 0.1 or 1 mg., or were insensitive. Somewhat similar figures were shown for the group of 10 patients with histologically proved ocular tuberculosis; 6, or 60 per cent, had cutaneous reactions to 0.001 mg.; 2, or 20 per cent, to 0.01 mg., and 2, or 20 per cent, to 0.1 mg. or less. These figures bear out the findings of Hart,[6] who tested the cutaneous reactivity of 1,030 patients with active systemic tuberculosis. He found that 13 per cent gave entirely negative reactions to 0.01 mg., and on testing with stronger doses he found that even with 100 mg. there was a residual error of 2.2 per cent in the test. These figures illustrate well the fallacy

TABLE 1.—*Hypersensitivity to Tuberculin of Patients with Ocular Tuberculosis*

		Sensitive to 0.001 Mg.		Sensitive to 0.01 Mg.		Sensitive Only to 0.1 or 1 Mg., or Insensitive	
Condition	Number	Number	Percentage	Number	Percentage	Number	Percentage
Clinical ocular tuberculosis	180	96	53.4	75	41.6	9	5
Histologically proved ocular tuberculosis	10	6	60.0	2	20.0	2	20

of the view that a high degree of cutaneous reactivity should be expected in patients with ocular tuberculosis and that a low degree of cutaneous reactivity is positive evidence against the presence of ocular tuberculosis. Unfortunately, one has no means of determining the degree of the ocular sensitivity, no knowledge of the influence of local disease on the ocular sensitivity and no information on the relationship of cutaneous and ocular sensitivity in patients with ocular tuberculosis.

METHODS AND RESULTS OF TREATMENT

In addition to tuberculin treatment, these patients all received such general hygienic treatment as was compatible with their social status. Certain selected patients were given special treatment, paracentesis, autohemotherapy, phototherapy and subconjunctival injections, as indicated. The results of these therapeutic measures, the use of tuberculin, general hygienic treatment and special treatment, will be discussed in order.

6. Hart, P. D.: The Value of Tuberculin Tests in Man, Medical Research Council, Special Report Series, no. 164, London, His Majesty's Stationery Office, 1932.

Treatment with Tuberculin.—Tuberculin, whether given on the basis of the perifocal concept or that of the desensitization concept, was at the onset of treatment administered in the same manner outlined in a previous communication. The initial dose was usually 0.0001 mg. of Denys bouillon filtrate, made and standardized in the laboratories of the Wilmer Ophthalmological Institute. The doses were increased in the usual manner and repeated every four days until a dose of 0.9 mg. was given. Thereafter injections were given once a week. The site of injection was watched for local reactions, and the eyes were constantly observed for any suggestion of focal reactions. With the occurrence of local reactions the dose was dropped three or four levels, and with any suggestion of focal reactions it was dropped drastically—eight levels—and increased only when local or focal reactions had disappeared, the effort being made consistently to keep the dose below the patient's individual point of reactivity.

TABLE 2.—*Results of Tuberculin Therapy in Patients Receiving Only One Course of Tuberculin*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis	23	10	12	1
Kerato-iritis	17	6	7	4
Scleritis	7	5	0	2
Posterior ocular tuberculosis	62	35	16	11
General uveitis	34	18	9	7
Total	143	74	44	25
Percentage	100	51.9	30.7	17.4

The most important difference in treatment between that based on what we have termed the perifocal concept and that based on what we have called the desensitization concept lies in the duration of treatment. The majority of these patients, 143 in all, received only one course of tuberculin. At the completion of the course, when the patient had received 90 mg. of tuberculin without reaction, treatment was abandoned. In the greater number of these patients the decision to abandon treatment was influenced by satisfaction with the clinical outcome or by conviction that tuberculin had accomplished all that could be expected. In other patients, for various reasons, it was impossible to continue treatment. The results in these patients, whose treatment with tuberculin falls under what we termed the perifocal concept of action, are shown in table 2. Seventy-four, or 51.9 per cent, showed apparent healing of the ocular lesions; 44, or 30.7 per cent, showed definite clinical improvement, while 25, or 17.4 per cent, were unimproved.

The method of treatment was radically different for the 32 patients for whom tuberculin was used on the basis of the desensitization concept

of action. After the dose of 90 mg. or the maximum dose that the patient could safely take was attained, injections were continued steadily over a period of at least eighteen months, even though the eye was apparently healed. For a few patients the dose was further increased, for one patient until the final dose of 500 mg. was reached, and this amount was given steadily at weekly intervals. For these patients the minimum period of treatment was two years, and treatment was then abandoned only if comparative nonreactivity of the skin had been attained and the condition of the eye was satisfactory. After treatment had been discontinued, the cutaneous reactivity was redetermined at the end of another three months. If the reaction was still negative, treatment was continued, but the cutaneous reactivity to tuberculin was determined regularly at three month intervals. With any return of cutaneous sensitivity, treatment with tuberculin was recommenced imme-

TABLE 3.—*Results of Tuberculin Therapy in Patients Treated Steadily Over a Minimum of Two Years*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis	2	2	0	0
Kerato-iritis	8	6	2	0
Scleritis	0	0	0	0
Posterior ocular tuberculosis	19	11	6	2
General uveitis	3	3	0	0
Total	32	22	8	2
Percentage	100	68.7	25	6.3

diately, again with a small dose at first as indicated by the degree of cutaneous reactivity. In several patients we have seen the recurrence of cutaneous sensitivity antedate the recurrence of activity in the eye. On this system the greater number of these patients have been followed for over five years, and in 4 patients with solitary tubercles of the choroid, tuberculous periphlebitis, tuberculous kerato-iritis and nodular iritis, respectively, the period of observation has now been over ten years. The results of treatment in this group of patients are shown in table 3. Twenty-two, or 68.7 per cent, appear totally healed. Eight, or 25 per cent, are definitely improved, with the disease under full control, but not as yet considered completely healed. Two patients showed no improvement. One of these patients had long drawn out generalized uveitis, which finally quieted but left a secondary cataract. The second patient's was distinctly an "uncompensated case," with severe, persistent scleritis and recurrent iritis. She was violently hypersensitive to tuberculin and showed repeated reactions to minute doses. She has never been able to take a larger dose than 1 mg. Over a period of six years, time and again we have been forced to abandon tuberculin therapy in this

case. This patient is included in the series only because from the first we purposed to treat her on the basis of the desensitization concept and despite repeated efforts have been unable to continue.

These two groups are not strictly comparable, owing to their difference in size, but they show a striking difference in the results obtained by the two methods of treatment. An obvious criticism is that the better results obtained by the treatment based on the desensitization concept are a reflection of a longer period of observation, that ocular tuberculosis tends naturally to healing and encapsulation and therefore the better results obtained in patients treated and observed over a minimum period of two years cannot fairly be attributed to the effect of tuberculin. In answer to this criticism we have clinical records for at least two years or over for 74 of the patients treated by one course of tuberculin, or under the perifocal concept. The results in this group are shown in table 4. Forty-two of these patients, or 56.7 per cent, still

TABLE 4.—*Results of Tuberculin Therapy with One Course of Treatment Only After Observation of Over Two Years*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis	11	8	2	1
Kerato-iritis	12	5	4	3
Scleritis	3	2	0	1
Choroiditis and retinitis	32	18	7	7
Uveitis	17	9	2	5
Total	74	42	15	17
Percentage	100	56.7	20.3	23

showed inactivity or healing of the original lesions; 15, or 20.3 per cent, continued to show improvement, while 17, or 23 per cent, showed no improvement, or had continuous relapses. These figures show no appreciable difference from the observation at the conclusion of the one course of tuberculin and indicate that the time element is not the factor responsible for the better results obtained in the group treated on the basis of the desensitization concept.

We have no figures of the results in a control series of patients with comparable ocular tuberculosis not treated with tuberculin. Hartig,[7] in his study of the effect of tuberculin treatment in ocular tuberculosis, withheld tuberculin for one year from a control group of 14 patients with this disease. In only 1 of these patients did he observe clinical improvement, while in the remaining 13 the disease remained unimproved, progressed or relapsed.

7. Hartig, F.: Ueber den gegenwärtigen Stand der Behandlung den Augentuberkulose, Ztschr. f. Augenh. **50**:79, 1923.

Table 5 shows the results of our total experience with tuberculin therapy in the 175 patients studied, whether treated on the basis of the perifocal concept of action or that of the desensitization concept. Ninety-six, or 54.7 per cent, on last observation appeared healed; 52, or 29.7 per cent, showed improvement, while in 27, or 15.4 per cent, the condition was unimproved or the eyes had progressed to blindness.

General and Climatic Treatment.—In treatment based on the desensitization concept of the use of tuberculin the obvious aims are to remove, by the use of tuberculin, the fatal tissue hypersensitivity responsible for the inflammatory, destructive phases of the lesion and, by other means, to promote the acquired immunity which favors the encapsulation and destruction of the bacteria. At the present time, unfortunately, there is no means of artificially increasing the immunity which to a greater or lesser degree follows any tuberculous infection. An attempt to this end has been made by the vaccination of both children and

TABLE 5.—*Total Results of Tuberculin Therapy in Patients with Ocular Tuberculosis*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis	25	12	12	1
Kerato-iritis	25	12	9	4
Scleritis	7	5	0	2
Posterior ocular tuberculosis	81	46	22	13
General uveitis	37	21	9	7
Total	175	96	52	27
Percentage	100	54.9	29.7	15.4

adults with BCG and killed virulent organisms.[8] At present, owing chiefly to the marked increase in the allergy which follows the injections, such vaccination is impossible in ocular tuberculosis. There are, therefore, only general means of promoting the acquired immunity.

The importance of rest, freedom from overstrain, cleanliness, fresh air, sunshine and an adequate diet in the treatment of any form of tuberculosis is recognized. For the patients reported on in this group, every effort has been made, so far as the social condition of the patients permitted, to meet these elementary aims. These simple hygienic regulations are of the greatest importance, and their proper maintenance, often with a change in environment, has apparently frequently marked the turning point in the course of the ocular disease. Nevertheless, in no case have we advocated sanatoriums or high altitude for the usual patient with ocular tuberculosis. The reasons for this are twofold. First, in the usual remote sanatorium for persons with tuberculosis it is impos-

8. Goodwin, T. C.: The Use of Protective Vaccines in Tuberculosis with Especial Reference to B.C.G., Internat. Clin. **2**:249, 1936.

sible for the patient to receive the careful observation of his ocular lesion necessary for successful tuberculin therapy. Second, in spite of Werdenberg's[9] contention that treatment with high altitude tends to lessen the dangers of untoward tuberculin reactions, we can see no reason why climatic treatment with high altitude should influence the course of ocular tuberculosis uncomplicated by active pulmonary lesions. Our experience in the coincidence of ocular tuberculosis with active pulmonary tuberculosis, which will be reported in detail elsewhere, does not agree with that of Werdenberg. While we do agree with the many investigators that ocular tuberculosis is invariably secondary to systemic tuberculosis and usually to infection of hilar glands, we have not been able to demonstrate constantly, either by physical or by roentgen ray examination, the pulmonary involvement invariably observed by Werdenberg. It appears either that we must be in error in our clinical diagnosis of ocular tuberculosis or that American radiologists must read as normal many of the 60 per cent of roentgenograms of the chest interpreted by Werdenberg as showing slight but definite evidences of tuberculosis of hilar glands or the lungs. In regard to treatment in sanatoriums and with high altitude, it is of some interest that 3 patients in this series came directly to Baltimore from sanatoriums for persons with tuberculosis where their ocular tuberculosis had progressed unfavorably. Two of these patients are now classed as healed, and 1 is classed as improved.

SPECIAL PROCEDURES

Paracentesis and Autohemotherapy.—These procedures are indicated especially for patients with the so-called "uncompensated" types of ocular tuberculosis, patients for whom Werdenberg[9] believes tuberculin is contraindicated. These patients usually have tuberculosis of the anterior part of the uvea, are extremely hypersensitive to tuberculin and show a rather intense, long-continuing inflammatory reaction, with a tendency to extension rather than limitation. They usually take tuberculin poorly, with local and sometimes focal reactions. Theoretically, in these patients one would imagine an excess of antigen, or tuberculoprotein, with spreading sensitization of the ocular tissues and an insufficient concentration of immune bodies in the ocular humors. These patients usually show an amazing improvement in the clinical picture, and may become "compensated" after paracentesis of the anterior chamber and drainage of the aqueous, a procedure which theoretically is accompanied by a large increase in immune antibodies in the reformed aqueous. We do not agree with Werdenberg that tuberculin is absolutely contraindicated in this group of patients, but we do agree that

9. Werdenberg, E.: Beurteilung und Behandlung der Augentuberkulose, Klin. Monatsbl. f. Augenh. (supp.) **94**:3, 1935.

it must be used in extremely small doses and with great caution until the intensity and extension of inflammation have been controlled by such a procedure as drainage of the aqueous or until the lesion has become compensated.

Autohemotherapy, or injection of the patient's own blood into the anterior chamber, the procedure advocated strongly by Schieck,[10] has, in our opinion, little advantage over simple drainage of the aqueous. The further concentration of antibodies incidental to the injection of such a small amount of whole blood into the anterior chamber must be slight. The beneficial effects reported for autohemotherapy may all fairly be attributed to the drainage of the aqueous incidental to this procedure. Our experience with simple drainage in these cases has been equally as satisfactory as when this was used with the added step of autohemotherapy, which always carries with it a slight additional risk of infection.

Phototherapy.—A. Ultraviolet Rays: Both general and local irradiation with ultraviolet rays have been used in the treatment of ocular tuberculosis. Duke-Elder [11] stated the belief that the effect of general irradiation of the body is due to complex photochemical and photoelectric changes, while the local effect is abiototic, with photochemical denaturation and coagulation of the protein. Duke-Elder,[11] Goulden,[12] Koeppe [13] and others have reported strikingly beneficial results in tuberculous iridocyclitis by general irradiation of the body, and in a few cases apparently good results with local irradiation of the diseased eye. On the other hand, Birch-Hirshfeld and Hoffman [14] expressed the opinion that ultraviolet irradiation is only an adjunct in the treatment of ocular tuberculosis.

We have used ultraviolet irradiation in a number of cases of tuberculosis of the anterior ocular segment. General irradiation with the use of a quartz mercury vapor lamp with doses just below or at the skin erythema dose produces a sense of well-being, improves the general tone of the patient and certainly does not aggravate the ocular lesion. On the other hand, we have observed no marked beneficial results

10. Schieck, F.: Die Behandlung von tuberkulösen Erkrankungen des vorderen Bulbusabschnitts durch Eigenblutinjektionen in die Vorderkammer, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **49**:183, 1932.

11. Duke-Elder, W. S.: Ultra-Violet Light in the Treatment of Ophthalmic Disease: I. General Phototherapy, Brit. J. Ophth. **12**:289, 1928; II. Local Phototherapy, ibid. **12**:353, 1928.

12. Goulden, C.: The Value of Ultra-Violet Rays, Tr. Ophth. Soc. U. Kingdom **46**:207, 1926.

13. Koeppe, L.: Die Diathermie und Lichtbehandlung des Auges, Leipzig, F. C. W. Vogel, 1919.

14. Birch-Hirschfeld, A., and Hoffman, W.: Die Lichtbehandlung in der Augenheilkunde, Berlin, Urban & Schwarzenberg, 1928.

which could reasonably be attributed to such treatment. Local irradiation with the use of the Birsch-Hirshfeld carbon lamp, with exposures of from thirty seconds to one minute, have been used almost as a routine in the treatment of deep scleritis and in some cases of kerato-iritis, due care being taken to protect the lens. In deep scleritis our general impression has been that the treatment has been of some value, but in no case have the results been striking. Local ultraviolet irradiation appeared to have little if any effect on corneal infiltrates or scars and did not appear to influence the course of iritis.

B. Roentgen Ray Treatment: The use of roentgen ray irradiation in ocular tuberculosis has been advocated by the German ophthalmologists, notably Stock,[15] Scheerer [16] and Werdenberg.[9] The most favorable results have been reported in nodular iritis, scleritis and keratitis. Stock reported poor results in cyclitis and choroiditis and no results in retinal periphlebitis. He reported the optimum dose to be 20 per cent of the skin erythema dose, and Scheerer, after first using a larger dose, now advocates a dose between 10 and 15 per cent of the skin erythema dose. Within from twelve to twenty-four hours after the irradiation of the eyes there ensues a more or less violent irritation, which disappears within three to four weeks, with marked improvement or even healing of the lesion. If necessary, the treatment may be repeated until a total of about 80 per cent of the skin erythema dose has been given. Scheerer recommends the use of a 0.5 mm. zinc and a 3 mm. brass filter, with a field measuring 4 by 4 cm. He believes the beneficial effects are due to degeneration of the lymphocytes and leukocytes in the lesion, while Werdenberg believes the good effects are due to the roentgen rays producing perifocal reactions about the lesion similar to those produced by tuberculin.

We have used roentgen ray therapy in only a few cases, and our experience has not been so fortunate as that just reported. The technic employed has been slightly different, but the dose is about the same. A 1 mm. aluminum filter and a 2 by 2 cm. field were used, and a calculated dose of 140 roentgens was given. Since 800 r. is about the skin erythema dose, this dose was apparently 17 per cent of the skin erythema dose, midway between the 15 per cent advocated by Scheerer and the 20 per cent recommended by Stock. We observed the same activation of the lesion reported by these authors but failed to observe cicatrization or healing of the lesion after the more or less violent primary reaction subsided. In fact, the primary activation appeared to aggravate the

15. Stock, W.: Strahlenbehandlung in der Augenheilkunde, Klin. Monatsbl. f. Augenh. **76**:542, 1926.

16. Scheerer, R.: Röntgenbestrahlung bei Uvealtuberkulose, Klin. Monatsbl. f. Augenh. **75**:27, 1925.

progress of the lesion, and we have abandoned roentgen ray treatment. Possibly, had a greater number of patients been treated or the treatment persevered in, the beneficial effects reported by others might have been observed. As it is, our limited experience has frankly been discouraging.

C. Radiotherapy: The use of radium for ophthalmic conditions and ocular tuberculosis has been studied, especially by Kumer and Sallman,[17] who have published a comprehensive monograph on the subject. They recommend radiotherapy, especially in tuberculous keratitis and iridocyclitis. In keratitis they note specifically the theoretical advantages of using the only slightly penetrating soft beta rays. However, since there are very few beta ray applicators in the world, they have used chiefly the gamma rays and a small amount of the hard beta rays. They used 100 mg. of the element in a 0.3 mm. platinum carrier which transmits 97 per cent of the gamma rays and 6 per cent of the hard beta rays (Fernau). In keratitis they have recommended direct or almost direct contact with the cornea and less than the skin erythema dose. In cases in which the condition is acute they used from 20 to 30 per cent of the skin erythema dose at intervals of two weeks. In tuberculous iridocyclitis they have employed either from 50 to 60 per cent of the skin erythema dose at one application or from 30 to 50 per cent of the skin erythema dose repeated at two week intervals and given at a distance from the eye. In general they reported good results in tuberculosis of the anterior ocular segment but noted that several patients were not helped and that in a few in whom the condition was active it was seriously aggravated. No effect was noted in juvenile retinal periphlebitis.

Our first experience with radiotherapy in ocular tuberculosis was confined to irradiation with the gamma rays, the dose being 1 gram minute at $\frac{1}{16}$ inch (0.2 cm.), approximately 50 per cent of the skin erythema dose. Only patients with kerato-iritis were treated. The results were unfortunate, although in no case was any permanent harm done. Definite activation of the uveal inflammation came on shortly after the treatment, persisted for some weeks and was not followed by a compensatory secondary phase of healing. It is true that there did appear to be some clearing of the corneal scars. Recently, through the agency of Dr. Curtis F. Burnam and the Howard A. Kelly Hospital, Baltimore, there has been available for use a beta ray applicator. The energy usually employed has been approximately 500 mg. of radon emanation in only the thinnest possible glass container, without a filter. This filter turns back any alpha rays and transmits practically all the soft beta rays and the gamma rays. The applicator is used in

17. Kumer, L., and Sallman, L.: Die Radiumbehandlung in der Augenheilkunde, Berlin, Julius Springer, 1929, p. 198.

direct or nearly direct contact for five seconds, the dose therefore being 2.5 gram seconds. This is repeated at two or three week intervals. This is approximately 50 per cent of the skin erythema dose of the beta rays and only about 4 per cent of the skin erythema dose of the gamma rays. The soft beta rays penetrate only from 2 to 3 mm. In the few patients treated to date we have observed not the slightest reaction and no activation of the uveal lesion. The results on the corneal infiltrates and scars have been remarkably good. There has resulted definite clearing of the cornea, and in one patient, who had a clear lens and a normal fundus, vision has improved from 20/100 to 20/50. Beta ray irradiation appears free from danger to the iris and lens and offers distinct promise in the treatment of corneal tuberculosis and in the clearing of corneal scars.

Subconjunctival Injections.—Subconjunctival injections of physiologic and hypertonic solution of sodium chloride have been used in a few patients with indolent inflammatory lesions in whom there was apparently little tendency to absorption and some mechanical procedure to hasten the process appeared in order. When daily subconjunctival injections of physiologic solution of sodium chloride were tolerated without reaction, the strength of the solution was gradually increased to a maximum of 3 per cent. These injections, with the attendant diffusion of fluids produced by different concentrations of sodium chloride beneath the conjunctiva and within the eye, appeared to promote the clearing of exudates and marginal corneal infiltration. Certainly no marked or dramatic change for the better followed their use, but there was likewise no activation of the lesion. While we are not unduly enthusiastic about the results obtained by subconjunctival injections of hypertonic solution of sodium chloride, on the whole they appear to be of some value in hastening absorption in cases in which the lesion is indolent.

COMMENT

In our early experience with tuberculin, influenced by the perifocal concept, the custom was to discontinue tuberculin when the dose of 9 cc. of no. 2 solution, or 90 mg., was reached. If the patient later showed a recurrence of ocular inflammation, tuberculin therapy was recommenced, the degree of cutaneous reactivity being again ascertained in order to determine the proper initial dose of tuberculin. At this time it was noted that the recurrence of the ocular inflammation was usually accompanied by a marked degree of cutaneous reactivity. It was also noted that a few patients under tuberculin therapy showed sudden changes and increases in the cutaneous sensitivity, which necessitated decreasing the therapeutic dose. This change in sensitivity often preceded a flare-up or recurrence of the ocular inflammation. In order to

ascertain if there was any relation between the apparent return or increase in the hypersensitivity to tuberculin and the recurrence of activity of ocular inflammation, the degree of cutaneous reactivity was determined at the end of a course of tuberculin therapy and redetermined at three month intervals thereafter. It was found that after tuberculin was discontinued there was often a steady return of the cutaneous reactivity, and this often preceded and apparently foretold the recurrence of ocular inflammation. In the light of this observation, tuberculin treatment was recommended on a return of the cutaneous sensitivity and before the expected ocular recurrence materialized. This procedure was sometimes unsuccessful, the ocular relapse occurring despite the treatment. We believed these recurrences could be explained on the grounds that there had been insufficient time to achieve any appreciable amount of ocular desensitization. We therefore approached the problem on the basis of the desensitization concept, the theory that tuberculin should be given with the idea of achieving tissue desensitization and must be steadily continued in order that this desensitization might be maintained and, as far as possible, made permanent. Our present ideas are that tuberculin must be given for a minimum of two years and discontinued then only if the skin has become almost totally nonreactive, and the ocular lesion has been entirely quiet and apparently encapsulated for a period of a year or longer. Even in such cases as these, whenever possible we redetermine the cutaneous sensitivity at three month periods and begin tuberculin therapy again with any sign of return of the cutaneous sensitivity, irrespective of the entire quiescence of the ocular lesion.

Occasionally patients are encountered who appear incapable of being desensitized. These patients are exquisitely sensitive to tuberculin and constantly show local, apparent systemic, and even focal, reactions to infinitesimal doses of tuberculin. Such patients may belong to the uncompensated type described by Werdenberg and be similar to one of the patients reported on in our series treated on the basis of the desensitization concept in whom treatment failed or may be patients with recurrent, brief inflammatory flare-ups in the eye, which are apparently related to the parenteral absorption of any amount of tuberculin. We have seen at least 2 patients of the last class. The only possible method of treating these patients with tuberculin would be to begin with a dose of 0.000001 mg., or even less, below their point of reactivity, whatever that may be, and increase the dose only with the patient under rigid observation.

The question of the influence of tuberculin treatment on recurrences of the ocular inflammation is difficult to analyze. We have lost track of almost half of the 143 patients treated on the basis of the perifocal

concept and therefore have no accurate figures for this group. Of the patients we have been able to follow, 36 are recorded as having shown one or more relapses, a total of 67 recurrences being noted. The majority of these recurrences were severe. The 32 patients treated on the basis of the desensitization concept, who have received tuberculin for a minimum of two years, have been more carefully followed. Twelve of these patients have shown recurrences. Ten have had one recurrence only, 1 had three minor recurrences and 1 a number of minor recurrences. With one exception, all the recurrences in this group were of low intensity and occurred rather early in the course of treatment, well before either systemic or ocular desensitization could have been accomplished. The severe recurrence occurred after four years of steady treatment. This recurrence appears to have been precipitated by the persistent injection of an excessive amount of tuberculin, to which the patient was definitely showing local cutaneous reactions. Happily, this patient recovered without residual damage after repeated paracentesis and the proper adjustment of the dosage of tuberculin. It is our distinct impression that the recurrences have been fewer and of less intensity in the patients treated on the basis of the desensitization concept than in the patients receiving only one course of tuberculin. Certainly we have seen no lost, phthisical eyes in the patients given prolonged tuberculin therapy, while such eyes have been observed in the group of patients who received what we now believe to have been inadequate treatment.

SUMMARY AND CONCLUSIONS

The results of tuberculin therapy and certain special forms of therapy in 175 patients with conditions diagnosed as various types of ocular tuberculosis are reported. One hundred and forty-three of these patients received only one course of tuberculin, being treated on the basis of what may be termed the perifocal concept of the action of tuberculin—the theory that tuberculin acts by causing small, subacute reactions about the ocular lesion, thus producing local immunity. Thirty-two patients received tuberculin more or less constantly over a minimum period of two years, being treated on the basis of what may be termed the "desensitization" concept—the theory that tuberculin acts by actually desensitizing the ocular tissues and so removing the factor responsible for the inflammatory and destructive phases of the tuberculous lesion. The outcome in the patients treated on the basis of the desensitization concept was decidedly better, both as to end-results and recurrences, than the results obtained in patients treated on the basis of the perifocal concept. From the theoretical, experimental and practical points of view, we believe the desensitization concept for the action and use of tuberculin

is correct and that tuberculin should be used in the treatment of ocular tuberculosis with the end-view of achieving and maintaining tissue desensitization.

General hygienic treatment is always indicated in ocular tuberculosis. Special procedures such as paracentesis, autohemotherapy, ultraviolet irradiation and subconjunctival injections are indicated in certain groups of patients. Our experience with roentgen ray therapy and gamma ray therapy has not been encouraging. Very encouraging results have been observed in our limited experience with beta ray radium therapy.

CORTICAL INNERVATION OF OCULAR MOVEMENTS IN THE HORIZONTAL PLANE

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A physiologist to whom ophthalmologists owe some of the best devised experiments on the mechanism of oculomotion recently expressed his opinion on the cortical innervation of ocular movement. This opinion, as expressed by him in various publications, is essentially in agreement with that of Duval and Laborde, although the latest anatomic researches show such a view to be untenable. Spiegel and Scala,[1] while apparently admitting the correctness of the latest data, viz., that the nucleus commissurae posterioris plays an important part in the mechanism of horizontal ocular movement, as does also a lateral nucleus in the globus pallidus as a tertiary center, stated: "There still exists much uncertainty concerning the cortical innervation of the ocular muscles." There seems to me to be some discrepancy in the reasoning of Spiegel and Scala, for, while admitting the uncertainty of the cortical innervation of the ocular muscles, they nevertheless have taken for granted, as shown by a full consideration of the results of their experiments, the existence of cortical centers for oculomotion and, further, that these stimuli are conducted by the capsula interna. As such controversy is a serious obstacle to progress in this field, I consider that the article written by Spiegel and Scala in clear and plausible style should not remain unanswered by those of the opposite camp.

As I have worked many years along purely physiologic lines (in the laboratories of Engelmann and Bowditch), Spiegel's close association with the physiologic side of the question and my own present association with anatomiconeurologic research need not exclude the possibility of our arrival at our common goal, i. e., a clear understanding of this most mysterious and evasive problem, the innervation of coordinated ocular movements.

This problem seems to me to appeal to the physiologist, the anatomist and the clinician, particularly ophthalmologists, otologists and neurologists, whose work brings them daily into contact with the practical side of the problem. At the same time it must be realized that any theory put forward by any one of these groups will be instantly criticized by the others. My own approach to the problem is along anatomico-

† Dr. Muskens died June 11, 1937.

1. Spiegel, E., and Scala, N. P.: The Cortical Innervation of Ocular Movements, Arch. Ophth. **16**:967 (Dec.) 1936.

physiologic lines in a research on forced movements. Since the mechanism operative in these forced movements in their three planes is the same as for ocular movements in their three planes (as shown in my recent book [2]), I am entitled to state: The permanent forced horizontal position, for instance, to the right, as seen in the human object and appearing in the experimental animal as horizontal conjugated deviation (with circular movement) to the right, occurs after cross-section of the left tractus vestibulomesencephalicus cruciatus, as well as after lesion of the right nucleus commissurae posterioris, the right nucleus lateralis globi pallidi and the connecting fiber tracts between the two latter structures. No permanent forced movement or position results in the human subject or in the experimental animal from a lesion of any other part of the central nervous system. It is particularly to be observed that this movement never follows lesions limited (by anatomic control) to the cortex cerebri or to the pyramidal tract, no matter how extensive these lesions are.[3]

Is one therefore in a position to deny the existence of any cortical influence on coordinated ocular movements? It must be admitted that one cannot go so far as this, but at least one can approach the question with an open mind, if somewhat cautiously. There can be no doubt that direct nervous connections exist between the pallidum and other structures, i. e., the neostriatum, the thalamus and the mesencephalon. Stimulation of the latter structures may induce ocular movements. As no direct fiber connection between the cortex and the pallidum is known, such stimuli must travel along an indirect path, i. e., the cortex, the thalamus, the pallidum, the nucleus commissurae posterioris and the tractus commissuromedullaris, thus demonstrating that ocular movements may result from stimulation of the cortex.

Such a question requires careful investigation and the task has been made much easier by such workers as D. Spiegel, who has elaborated a skilful method of examining vertical movements. It is interesting to remember that this very point baffled such an eminent worker as R. Russell, even with Hughlings Jackson as his advisor![4]

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2. Muskens, L. J. J.: Das supravestibuläre System, Amsterdam, N. V. Noord-Hollandsche Uitgevers Maatschappij, 1934, chaps. 2, 9, 11 and 16.

3. Muskens,[2] chap. 22.

4. Spiegel and Teschler's conclusion (Arch. f. d. ges. Physiol. **222**:370, 1929) that the vestibular nuclei are a station by which cortico-oculomotor innervation has to pass seems to me not warranted. Not only do anatomists know of no fiber relation between the pyramidal tract and the vestibular nuclei, but another view seems more acceptable. According to the modern notions, the vestibular nuclei, with their ascending—and, from the commissural nuclei, descending—connections produce the necessary background on which striatal, tectal or cortical (by some roundabout way) innervation has to play. This well attuned background lacking no properly coordinated horizontal ocular movements can occur.

When one recalls the work of Luciani and Tamburini, who regarded ocular movements following cortical faradization as being dependent on escape of current through the brain tissue [5] and, further, that clinicians and anatomists profess complete ignorance of any cortical tracts which convey oculomotor stimuli, it is obvious that this is a matter about which one must be very doubtful. That the secondary and tertiary supravestibular nuclei, which certainly play a part in the production of coordinated horizontal ocular movements, may receive stimuli in some more or less complicated way via collateral fiber tracts from neostriatal and cortical structures (Monakow) no one would deny.

But the point at issue is: Can it be proved beyond doubt that there is a direct fiber tract from some cortical area by way of the pyramidal tract and the capsula interna which, either on cross-section or on stimulation, disturbs or induces coordinated ocular movements? In my opinion, this cannot be proved, and on this point I differ from Spiegel and Riley, who, with others, have affirmed unhesitatingly that there is such a tract.

How did the idea that the pyramidal tract is associated with oculomotion innervation originate? Starting from the assumption that all voluntary movements are innervated from the pyramidal tract, Dejerine submitted the possibility that oculomotion follows the usual rule of voluntary innervation. Windler suggested that the aberrant pyramidal tract may be responsible, while Spiller discussed the matter in the same light but in more detail. In this way the idea took firm root, although proof was lacking, and the fact that the oculomotor mechanism exists in lower animals in which the pyramidal tract is not yet elaborated was overlooked, as was also the fact that oculomotion in three planes in those lower animals was part and parcel of the general body locomotion. At the same time, it must be pointed out that Leyton and Sherrington found that the mechanism of oculomotion is differently organized from that of voluntary muscular movements, while Wernicke considered ocular movements to be probably reflex, the impulse alone being voluntary.[6]

5. If one compares the method used by Ken Taga (Arch. f. d. ges. Physiol. **223**:117, 1929), who got better results when he stimulated a locality nearer the pallidum, viz., the capsula interna, with the old experiments of François Franck, it appears as if the Italian authors hit not far beside the mark. When judging the influence of the cross-section of the posterior commissure on the coordinated ocular movements it is well to recall that according to Godlowski's stimulation experiments Ganser's ventral commissure might compensate for the loss of function of the dorsal (i. e., posterior) commissure.

6. In regard to the reflex gaze, which is often present in cases of horizontal conjugate deviation of the head and eyes, caused by destruction of the globus pallidus (Muskens, L. J. J.: Das supravestibuläre System, Amsterdam, N. V. Noord-Hollandische Uitgevers Maatschappij, 1934, chap. 16) and its experimental

(Footnote continued on next page)

The clinician is well aware that in case of hemiplegia in which the capsula interna is completely destroyed ocular motion is rarely affected unless the striatum (more accurately, the globus pallidus) is involved (Prévost). And in a case of conjugate deviation with persistence of the simple vestibular reflexes, one is entitled to diagnose an intact fasciculus longitudinalis posterior and a lesion of the pallidum or of its connection with the commissural nuclei. Further, in experiment, as in disease, nystagmus, with a slow jerk to the right, is the first step toward conjugate deviation to the right,[7] and the notion had to be given up that the quick jerk of supravestibular nystagmus originates in the cortex (Bauer and Leifler, de Kleyn, Wilson and Pike, and Ivy). All this will be found in full accordance with the modern notions.

There is at least enough basis for the argument that the oculomotor mechanism ought to be regarded as a phenomenon sui generis. Without regard to other (at least pyramidal tract) motor mechanisms, I submit that the proper method of approaching the question of the mechanism of oculomotion is consideration of anatomicophysiologic data (two fundamental experiments are described in my work[8]) which are based on the (controlling) experience of twenty-three years of physiologic, anatomic and clinical work, which experience serves excellently as a foundation on which a theory or doctrine regarding the innervation of coordinated oculomotion could be built.

If in a larger number of lesions of the brain stem involving division of tracts or disturbance of nodal points up to the pallidum but not farther the coordinated movement is found to be disturbed, is it not better to keep firmly to known physiologic facts rather than to sail away before the capricious winds of fantasy?

Further, should it not be remembered that such authors as Leidler[9] the otologist, Nordmann[10] the ophthalmologist and Godlowski[11] the

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corollary, Graham Brown's orientation reflex (Arch. néerl. de physiol. **7**:344, 1922), there can be little doubt left about its localization. For Graham Brown found the reflex present as long as the posterior commissure and the fasciculus longitudinalis posterior was intact, and absent after cross-section through the corpora quadrigemina posteriora. As far as I can see, this result is in entire concordance with the new doctrine, according to which the infracommissural nuclei are the nodal points for reflex locomotion and oculomotion.

7. For this reason it seems now to be generally admitted that the slow jerk is the main feature of the syndrome, the past pointing being directed in the same sense. Hence I urged—till now in vain—that the direction of the nystagmus should be named after the direction of the slow jerk.

8. Muskens,[2] chap. 9.

9. Leidler, R.: Fragen der Lokalisation innerhalb des zentralen vestibular Systems, Monatschr. f. Ohrenh. **70**:267, 472, 544, 725, 801 and 951, 1936.

10. Nordmann: Personal communication to the author.

11. Godlowski: Podkowa osradki ipojzzenia I, Krakow, 1936.

neurologist have admitted that these novel views are far preferable to the antiquated ones already referred to? Of course, the outlook would be different if the two fundamental experiments, the bedrock from which I started, were proved to allow of other interpretation. This is the weakest link in the chain, and if anatomists and physiologists like Spiegel and his colleagues would either confirm these comparatively simple experiments (in which osmium-stained control preparations were used) or demonstrate a fallacy in them, the first step toward the final solution of this irritating riddle of oculomotion would be made.

DETACHMENT OF THE RETINA

OPERATIVE RESULTS IN ONE HUNDRED AND SIXTY-FOUR CASES

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AND

JOHN P. MACNIE, M.D.

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In the 164 cases of retinal detachment analyzed in this paper the eyes were operated on in the Institute of Ophthalmology of the Presbyterian Hospital, New York, between April 1, 1934, and Jan. 1, 1937. The total number of eyes operated on was 171. No attempt was made to select the cases, as it is still our feeling that almost all eyes in which every possibility of spontaneous cure has been eliminated should be offered a chance of benefit by surgical intervention. This attitude has led us to attempt treatment in many so-called hopeless cases. The results in this type of case were, on the whole, disappointing, yet surprising recoveries occurred sufficiently often to renew our interest in these very unfavorable cases. Some form of diathermy was used in practically all the cases, with an occasional minor variation in operative technic.

GENERAL STATISTICS

Sex.—In this series of 164 patients 67 per cent were males and 33 per cent were females. This difference is at least partially accounted for by the higher incidence of frank trauma in the males. A definite history of trauma was obtained in 21 per cent of the males and in only 9 per cent of the females.

Age.—The youngest patient was 6 years of age, and the oldest 79 years. In each decade between the ages of 20 and 60 there were but slight differences in the number of cases. There were 7 patients under 10 years of age in this group. Four of the adults were more than 70 years old.

Trauma.—In this series of 171 eyes a positive history of trauma was obtained in 29 cases (17 per cent). We were careful to include in this group only cases in which there was a definite causal relationship between the injury and the onset of the detachment. In 23 other cases (13 per cent) the accident was considered to be at least a contributory

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factor. A history of trauma was obtained from only 10 per cent of the patients with high myopia, while 23 per cent of those with low refractive errors (exclusive of aphakic patients) gave a history of injury. A cure was effected in 62 per cent of the cases in which trauma was definitely established as the causative factor.

OCULAR FINDINGS

In the cases of unilateral retinal detachment no preference was found as to the eye involved. If this series is combined with the one we [1] reported in 1934, there are 314 cases. In this group the right eye alone was affected 131 times and the left eye alone 128 times. Both eyes were involved in 55 instances, thus making the detachment bilateral in 17.8 per cent of all the cases. The frequency with which the fellow eye sooner or later becomes involved is striking, and in all probability if this series is followed for a longer time this percentage will rise. This fact should serve as an added stimulus to ophthalmologists to conserve all possible vision in every case of retinal detachment. Analysis of the 31 cases of bilateral retinal detachment in the present series showed 15 of the patients to have high myopia (requiring a sphere of over −6.00 D.) and 7 to be aphakic.

Refraction.—Analysis of the refractive error in this series of patients revealed myopia to be present in 53.8 per cent. In the series reported on in 1934, 66.6 per cent were myopes. These figures, along with those reported by others show that some degree of myopia exists in at least 50 per cent of all cases of retinal detachment. It is interesting to note that myopia of 6 D. or over was present in 30.4 per cent of this group. Of the 13 aphakic patients in this series, 5 had high myopia.

Muscular Imbalance.—In the present series no annoying postoperative diplopia resulted from the severance and reattachment of the rectus muscles. In a few instances a small amount of deviation was detected on routine postoperative tests of muscles, which, however, was usually insufficient to require a prismatic correction. This fact clearly indicates that one need have no fear about detaching one or more of the rectus muscles whenever such a procedure is necessary for a good operative exposure.

Intra-Ocular Tension.—As was suggested in 1934, hypotony is a grave prognostic sign. In the present series there were 17 instances in which the preoperative intra-ocular tension was below 10 mm. of mercury (Schiötz). Operations in these 17 cases resulted in 15 failures.

1. Dunnington, J. H., and Macnie, J. P.: Detachment of the Retina: Operative Results in One Hundred and Fifty Cases, Arch. Ophth. **13**:191-200 (Feb.) 1935.

It was noted in our previous article that in 5 instances in which the preoperative intra-ocular tension was less than 6 mm. of mercury no cures were effected. Hypotony is usually found along with other unfavorable signs, e. g., extensive detachment, but it is occasionally encountered in a person with detachment who appears in all other respects to be a good operative risk. It is therefore sugggested that the intra-ocular tension be recorded preoperatively in every case of retinal detachment.

Increased intra-ocular tension occasionally accompanies a serous detachment of the retina, as was noted in 6 of our cases. In no instance was there a return of the glaucoma after the operation for the retinal detachment. The retina was satisfactorily reattached in 3 of these 6 cases.

Changes in the Crystalline Lens.—Complete opacification of the lens developed postoperatively in 7 of the cases in which the results of operation were unsuccessful. Lenticular opacities were frequently found preoperatively in the patients over 50 years of age, yet an increase occurred postoperatively in only 1 instance in which satisfactory reattachment was obtained. The rarity of a postoperative increase in the lenticular changes seems to indicate that the operation itself has no appreciable effect on the status of the crystalline lens.

Changes in the Field of Vision.—In all cases careful perimetric examinations were made before operation. The changes in the field were found to correspond closely with the area of the detachment. After operation, in certain cases a defect in the field persisted which seemed to correspond closely to the areas of damage to the retina and choroid produced by the operation.

In those cases in which the detachment involved the macular region, replacement of the retina was not followed by a complete return of central vision.

From an analysis of these cases we still feel as we did in 1934 that if careful perimetric tests are done almost all eyes will show some impairment of function. Furthermore, it is our opinion that the degree of reaction necessary for cure in itself produces some reduction in the sensitivity of the retina.

RELATION OF VARIOUS FACTORS TO THE OPERATIVE RESULT

Time of Onset.—The series was studied to determine the relationship between the time of onset and the operative result. When those cases in which approximately one half of the retina was detached were considered it was found that the duration of the detachment had little, if any, influence on the result. In this group cure was effected in approximately two thirds of those patients operated on within one month after the onset. In similar conditions of over three months'

duration cure was also effected in two thirds of the cases. Attention should be called to the difficulty in determining the exact date of the occurrence of the detachment. Only those cases were included in this study in which this date could be established with reasonable certainty.

Among the patients with approximately one half of the retina detached there were 7 in whom the detachment had existed for at least a year. In 4 of these cases a cure resulted after operation; the longest interval between the onset of the detachment and a successful operative result was four years. In the group with more extensive detachment of the retina (three fourths or more of the retina) a favorable result (cure or improvement) was obtained in approximately one fourth of those patients operated on within one month after the onset. The same percentage of cures was obtained in the cases of extensive detachment of over three months' duration.

Site of Detachment.—In those patients with approximately one half of the retina detached the inferior portion was affected in 50 per cent. Treatment confined to this region was successful in 74 per cent.

TABLE 1.—*Relation of Extent of Detachment to the Operative Result*

Ninety-Nine Eyes with One Half or Less of the Retina Detached		Seventy-Two Eyes with Three Quarters or More of the Retina Detached	
Result	Percentage	Result	Percentage
Cure	64.6	Cure	11.1
Improvement	4.1	Improvement	13.9
Failure	31.3	Failure	75.0

The temporal half was detached in 31 per cent, with involvement of the nasal or the superior parts in approximately 9 per cent. In these cases a successful result was obtained in between 55 per cent and 60 per cent. The figures just given illustrate that the location of the detachment has but little bearing on the outcome.

Extent of Detachment.—In this analysis an attempt was made to determine what rôle the extent of the detachment played in the operative result. This is strikingly shown in table 1.

The percentage of cures in those cases of detachment involving one half or less of the retina is most encouraging, but equally heartening is the fact that 25 per cent of the patients with extensive detachment can be benefited by operation.

Type of Detachment.—In this series the detachment was considered to be bullous in 65 per cent of the cases, mixed in 17 per cent and flat in 17 per cent.

Of those patients with a bullous detachment involving not more than one half of the retina, approximately 65 per cent were benefited by

operation. Among 18 instances in which one half or less of the retina was very slightly elevated, i. e., flatly detached, a cure was obtained in 14 (77.7 per cent).

The term mixed was used for those extensive detachments in which a bullous separation was accompanied by a slight elevation of a different portion of the retina. Consequently in this group the operative results were more unfavorable, failure occurring in 57 per cent of the cases.

Holes.—An attempt was made to seal all existing holes by concentrating the treatment in these areas, but, as was advocated in 1934, the entire area of the detachment was treated whenever feasible. The thought back of such widespread treatment is the desire to create an extensive area of adhesive choroiditis, which we feel insures a more permanent result. The consensus seems to be that the so-called "blind" operations are not often successful. However, a study of these cases shows that 54 per cent of the patients in whom no retinal holes were located preoperatively were benefited, while of those in whom one or more retinal holes were found before operation 48 per cent were either cured or improved. Analysis of the cases in which large or multiple holes or extensive disinsertions were found shows that a favorable result was obtained in 46 per cent. Among patients in whom only a single small hole was seen preoperatively a satisfactory result was obtained in 60 per cent.

DESCRIPTION OF OPERATIVE METHOD

Anesthesia induced by tribromethanol in amylene hydrate supplemented with ether was used during the majority of the operations. It is felt that there was little difference in the postoperative course between general and local anesthesia.

In all the operative methods the sclera was exposed over the extent of the detachment, and in most of the operations one or two of the rectus muscles were severed. In a few instances satisfactory exposure was obtained by retracting the muscles without detaching them.

In the majority of the operations diathermy was used. In approximately the first half of the period covered by this report (April 1, 1934, to Jan. 1, 1937) the Walker type of platinum needles was used with the Walker instrument. These were inserted into the sclera over the area corresponding to that of the detachment. The current was adjusted so that penetration took place with little pressure within a second or two after the contact was made. After all the needles were inserted they were withdrawn, the subretinal fluid being allowed to escape.

In the summer of 1935 a modification by Unsworth and Larkin of the Lacarrère electrodiafaco instrument was adopted in place of the platinum needles. With this method a wire of adjustable length can be used, permitting either surface application or penetration. The entire surface of the sclera corresponding to the area of the detachment was treated. From thirty to fifty applications were used, depending on the extent of the detachment. In some instances all the applications were perforating, while in others treatment was confined to surface coagulation except for a few final punctures to permit escape of the subretinal fluid.

Multiple trephining with chemical cauterization of the choroid (the method of Guist and Lindner) was used in but 10 instances in the early part of this series. This method has been discarded except when used in combination with one of the methods of diathermy, at which time one or more trephine openings are made, treated chemically and opened with a punctum dilator to insure better drainage of the subretinal fluid. The combined method was used in 49 operations.

Dr. Clifford Walker treated one patient with diathermy and injection of a 3 per cent solution of sodium hydroxide into the region of the macula and another by electrolysis. In each case cure resulted. One patient was operated on with the thermophore heated to 170 F. and applied for one minute to eight points in the inferior third of the globe between 12 and 17 mm. posterior to the limbus. One trephine opening 14 mm. posterior to the limbus below was made for drainage. This patient was cured by operation.

Postoperative care consisted in confinement to bed for approximately two weeks. During this time both eyes were bandaged and dressed every forty-eight hours, with the instillation of a 1 per cent solution of atropine sulfate into the affected eye. A gradual increase in activity was then allowed, and the patient was discharged from the hospital in about three weeks.

ANALYSIS OF RESULTS

In the entire group of 171 eyes operated on the detachment was either cured or improved in 50.3 per cent (cured in 42.1 per cent and improved in 8.2 per cent), while in 49.7 per cent failure resulted. The eyes were considered cured when the retina was completely reattached and there was enlargement of the field of vision. The "improved" eyes were those in which the field of vision became enlarged yet some peripheral detachment persisted or else the retina was in place but the field of vision was not materially enlarged.

In this series 23 patients had more than 1 operation. In 2 instances 4 operations were done. In one of these cases complete reattachment was eventually secured, while in the other the operations were unsuccessful. Of the 4 patients who had 3 operations, only 1 was benefited (improved). Twenty-seven eyes were operated on twice. Of these, 9 were cured and 5 improved.

An analysis of the 72 cases in which the eyes were cured showed that only 2 had a reduction in visual acuity following the operation. In one of these vision decreased from 20/200 to 7/200, and in the other it decreased from 20/50 to 20/50—. It remained unchanged in 5 cases and was improved in all the others. In 38 instances postoperative visual acuity of 20/50 or better was obtained.

Failures.—As in the study reported in the previous paper, the records of the cases in which the operation was unsuccessful (85) were analyzed, and an attempt was made to determine the cause of failure. In 12 of these cases no cause could be ascertained. The following unfavorable conditions were frequently encountered:

(*A*) Age: The relation between the age of the patient and the operative result is strikingly shown in table 2.

It is interesting to note that none of the 4 patients over 70 were benefited by operation.

(*B*) High Myopia: Among those patients with myopia of over 6 D. only 48 per cent were benefited by operation, while in the group with emmetropia or low hyperopia operation was successful in 72 per cent.

(*C*) Aphakia: Among the 13 patients with aphakia only 2 were benefited by operation. In each instance the detachment occurred after the removal of the lens. In 7 of these cases the operation was extraction of a hard cataract. Detachment followed discission for secondary cataract in 4 cases and came on in 1 case after needling for a congenital cataract and in 1 case after removal of a dislocated lens. Nine of the 11 patients in the group with aphakia in whom operation resulted in failure had separation of three fourths or more of the retina. In the 2 who were benefited approximately one half of the retina was involved.

TABLE 2.—*Relation Between Age and the Operative Result*

Age, Years	Cases Resulting in Cure, Percentage	Cases Resulting in Improvement, Percentage	Cases Resulting in Failure, Percentage
Up to 50	50.0	9.2	40.8
50 or over	28.6	6.3	65.1

(*D*) Hypotony: Operation helped only 11.7 per cent of those patients showing a preoperative intra-ocular tension of less than 10 mm. of mercury (Schiötz).

(*E*) Extensive Detachment: There was detachment of at least three fourths of the retina in 52.9 per cent of all the patients in whom operation resulted in failure.

(*F*) Multiple or Very Large Tears: There were multiple holes, large tears or extensive disinsertions in 38.8 per cent of the patients operated on unsuccessfully.

(*G*) Changes in the Choroid and Retina: As might be expected, degenerative changes in the choroid and retina were more frequently encountered among the cases in which operation resulted in failure.

Hemorrhage into the vitreous occurred postoperatively in 11 cases, and in 9 of these the retina remained detached. In 7 cases the retina was in position on the patient's discharge from the hospital but subsequently became redetached. The longest interval at which this occurred was two months. In this series 2 eyes came to enucleation. Severe

iridocyclitis necessitated the removal of one, while the other was lost as the result of endophthalmitis. In both instances one or more trephine openings were made in the sclera at the time of operation.

SUMMARY

In this series of 164 patients with retinal detachment 67 per cent were males and 33 per cent were females. The ages varied from 6 to 79 years.

A definite history of trauma was obtained in 17 per cent, while an additional 13 per cent gave an indefinite history of injury. A cure was effected in 62 per cent of the patients with a positive history of trauma.

Both eyes were found to be involved in 31 patients in the series.

Some degree of myopia was present in 53.8 per cent of the patients, 30.4 per cent of the group having myopia of at least 6 D.

Hypotony was found to be a grave prognostic sign.

In the patients having one half or less of the retina detached, improvement was obtained in 68.5 per cent, while in those with three fourths or more of the retina involved only 25 per cent were helped by operation.

The inferior portion of the retina was found to be the site of the detachment in 50 per cent of all the cases.

Those eyes in which no retinal holes were found responded to operation as well as those in which one or more retinal holes were located preoperatively. Widespread treatment of the affected area was used in all the cases.

In this group of 171 eyes the results of operation were: cure in 42.1 per cent, improvement in 8.2 per cent and failure in 49.7 per cent. Advanced age, high myopia, aphakia, hypotony, extensive detachment and multiple or large tears were frequently encountered among the cases in which operation resulted in failure.

INTRA-OCULAR NEUROFIBROMA

REPORT OF A CASE

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Neurofibroma within the eye has been so infrequently reported that every instance in which it occurs should be recorded. For this reason I wish to report the following case.

REPORT OF CASE

History.—B. D., a youth 21 years of age, was seen by Dr. J. H. Roth, of Kankakee, Ill., on Oct. 11, 1932. He gave a history of failing vision in the left eye, which had begun two years before, and of total loss of vision for the past year. Three weeks before examination the left eye became red and painful, and there was considerable tearing. The pain kept him awake at night. A general examination was not made.

Examination revealed the left eye deeply injected, the cornea hazy and the pupil widely and symmetrically dilated. There was no perception or projection of light. Vision of the right eye was 1.5—3. On October 20 the cornea was clearer, and it was thought a tumor could be seen within the eye on the temporal side. The tension was 59 mm. as measured by the Schiötz tonometer. The Wassermann test of the blood and urinalysis gave negative results. The following day, on examination in consultation with Dr. Harry Gradle of Chicago, a large tumor was visible on the temporal side of the fundus. Paracentesis of the anterior chamber to facilitate a better view of the fundus was advised. This procedure resulted in a hemorrhage into the anterior chamber, which cleared in two days, whereupon the tumor was again seen. The eye was enucleated on October 27.

Three and a half years later, attempts to find the patient for follow-up examinations were unsuccessful, as he had moved from the community.

Pathologic Examination.—The gross appearance of the bulb was normal. On transillumination a tumor was visible in the temporal quadrant.

After fixation the bulb was sectioned serially, each section being cut at 13 to 22 microns. Every tenth section was stained by Salzmann's hematoxylin-eosin technic, and other sections were stained with specific stains.

Microscopic Study.—The cornea was normal. Section 270 showed a subconjunctival paracentesis opening into which the angular line of the iris barely presented, the space remaining between the lips of the wounds and the conjunctiva being filled with a serofibrinous exudate similar to that filling the anterior chamber. This opening appeared to measure 0.75 mm. horizontally.

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Read before the Eye, Ear, Nose and Throat Section of the Houston Academy of Medicine, May 20, 1937.

The peripheral fourth of the iris was adherent to the back of the cornea. There was a slight tendency for the iris pigment to curve onto the anterior surface of the iris at the pupillary border. The lens showed occasional small round droplets in the posterior cortex and was otherwise distorted, probably from fixing and sectioning.

The ciliary body was markedly thinned, and its processes were small and sparse.

The retina had been totally detached. The subretinal space was filled with a serum-like exudate and was intruded on by a tumor which had choroidal tissue reflected over it.

This tumor occupied a central position about 0.3 mm. temporal to the disk; it projected axially 10 mm. and was 14 mm. across in its broadest expanse as measured on the stained section. The base of the tumor adjoined the sclera, the outer layers of the tumor being more compact. It showed moderate vascularization, the walls of the vessels consisting of a single layer of endothelium. These were not, however, true blood vessels, as no red blood cells were seen in most of them. The cells of the tumor were spindle shaped and were fairly regularly arranged, usually in columns or palisades, the long axis of the cells being perpendicular to the direction of the columns, many of which were thrown into irregular folds and whorls (fig. 1). The space between the columns contained an undifferentiated cytoplasm, no cellular membranes being distinguishable. Occasionally in these areas a spindle-shaped nucleus was seen, with its axis more or less parallel to the palisades. No mitotic figures were seen, and no melanotic cells were present except in the outermost layer adjoining the sclera or choroid, where the usual chromatophores were visible.

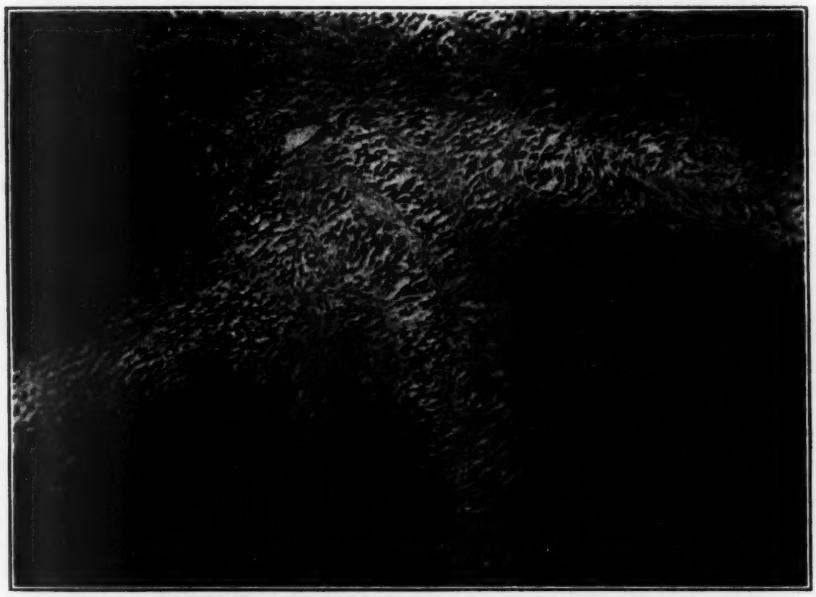

Fig. 1.—High power photomicrograph, showing palisading of cells, with cystic spaces and intercolumnar cells.

Serial section 180 showed, just outside the sclera, a cross-section of an artery, a large vein, a small vein and a nerve. The last was no longer characteristic

but was composed of a central area containing many nuclei, most of which were spindle shaped, having no regular arrangement and lacking any distinct separation from the surrounding tissue; these cells resembled the centrally lying spindle cells and faded out into a nearly normal appearing sheath of connective tissue

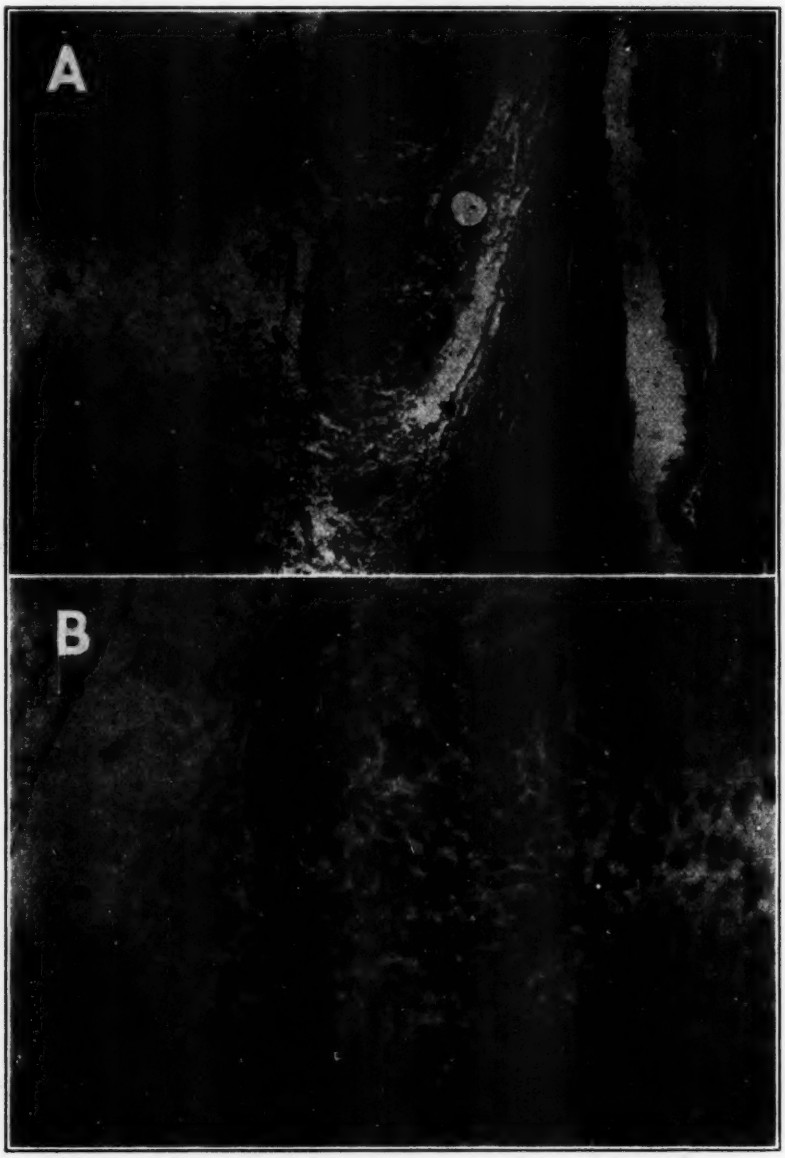

Fig. 2.—*A*, low power field, showing an artery and small vein and a cross-section of an abnormal ciliary nerve. *B*, high power view of the ciliary nerve shown in cross-section.

(fig. 2). Unfortunately, the sclera had been torn in the process of embedding and reembedding, because of air bubbles and sectioning, so that this group of struc-

tures could not be followed through the sclera. It is my opinion, however, that this is a part of intra-ocular formation which outside the eyeball had become plexiform in character. Other posterior ciliary nerves were seen in cross-section and longitudinally, but none showed this striking variation from the normal.

The optic nerve was normal; the retina had been torn from it during fixation.

A Perdrau stain of section 154 revealed a more or less dense net of argyrophilic fibers running mainly in a direction parallel to the palisades, where this formation was distinct, and elsewhere intermingling in a lacelike manner to form the supporting network. These fibers had processes extending like footplates to the walls of the vacuoles.

A diagnosis of intra-ocular perineurial fibroma, with secondary glaucoma and serous detachment of the retina, was made.

COMMENT

This neurofibroma, or perineurial fibroma, seems to have arisen from a ciliary nerve and showed changes similar to the plexiform neuroma, of which there are numerous reports. This single nonmalignant tumor of the nervous system has been named perineurial fibroblastoma because of its microscopic structure. However, the title which I have used is still the one in common usage. Penfield [1] defined a perineurial fibroblastoma as "an encapsulated tumor which arises from the sheaths of spinal nerve roots, cranial nerves, and peripheral nerves. It appears usually as a solitary tumor, much more often in a central location than a peripheral one. Tumors of the acoustic and spinal nerve roots far outnumber all others." His description of the microscopic details resembles closely that already given. He further stated that this tumor does not metastasize but may recur locally unless the segment of attached nerve is also removed. Its growth is usually slow. Sarcomatous degeneration has been reported but infrequently. Weil's [2] description is essentially the same as the foregoing one. He stated that formation of cysts is another feature of this tumor.

It may well be that the spaces described in my case are cystic changes rather than blood vessels. Indeed they were so interpreted for me by my colleague, Dr. Paul C. Bucy,[3] the neurologist. He stated:

> This is a typical neurofibroma such as one sees on peripheral nerves or on the acoustic nerve, in spite of its unusual location. Sections impregnated by Perdrau's silver stain for reticulin are not entirely satisfactory but do reveal many fibers of reticulin among the spindle-shaped cells of the tumor.

The pathologic observations on a leukosarcoma of the iris by Zentmayer [4] were somewhat similar to those in the case reported here, as

1. Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 968.
2. Weil, A.: A Textbook of Neuropathology, Philadelphia, Lea & Febiger, 1933, p. 276.
3. Bucy, P. C.: Personal communication to the author.
4. Zentmayer, W.: Primary Sarcoma of the Iris, Removed by Iridectomy, Tr. Am. Ophth. Soc. **28**:109, 1930.

the cells in his case were spindle shaped, arranged in bundles curling this way and that and had large blood vessels, the walls of which consisted only of endothelium. No mention was made of the presence of mitotic figures, and no silver stains were made. Roentgen therapy was utilized, and there was no recurrence of the tumor after three and a half years.

Two neurofibromas in one eye were described by Callender and Thigpen [5] in 1930. In their cases the tumors were "made up of spindle-shaped cells often arranged in whorls, occasionally having a palisade arrangement around a hyalin or fibrillar matrix. The ciliary tumor is somewhat more vascular and more cellular than the posterior one." My examination of a partial section of this eye (loaned by the Army Medical Museum) showed the tumor of the ciliary body to be somewhat similar to that in my case but with not nearly as characteristic an arrangement of the cells.

A flat neurofibroma of the choroid described by Freeman [6] in 1934 originated from the perivascular nerve plexus; it was not nodular in type, nor did its cells show palisading.

A case of melanosis of the uvea and melanoma of the iris, associated with generalized neurofibromatosis, has been described by Goldstein and Wexler.[7] This condition is an entirely separate one, more likely belonging in that large group of conditions known as von Recklinghausen's disease, whereas the tumor which I have described belongs with the solitary neurofibromas.

Papoleczy [8] described an intrabulbar and retrobulbar neurinoma arising from a posterior ciliary nerve, and Nitsch [9] reported two cases of neurofibroma of the eye. These reports were not available for this paper.

The discussion of the neurofibromas of the orbit has been purposely left out of this paper, but in a case of such a tumor, a recently reported one by Motto,[10] the structure was shown to be similar to that of the

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5. Callender, G. R., and Thigpen, C. A.: Two Neurofibromas in One Eye, Am. J. Ophth. **13**:121, 1930.

6. Freeman, D.: Neurofibroma of the Choroid, Arch. Ophth. **11**:641 (April) 1934.

7. Goldstein, I., and Wexler, D.: Melanosis Uveae and Melanoma of Iris in Neurofibromatosis (Recklinghausen), Arch. Ophth. **3**:288 (March) 1930.

8. Papoleczy, F.: Intrabulbar and Retrobulbar Neurinoma Arising from Posterior Ciliary Nerve, Arch. f. Ophth. **128**:326, 1932.

9. Nitsch, M.: Neurofibromas of the Eye: Two Cases, Ztschr. f. Augenh. **69**:117, 1929.

10. Motto, M. Paul: Neurofibroma of the Orbit, Arch. Ophth. **17**:340 (Feb.) 1937.

tumor occurring within the eye in the case reported here. The tumor in Motto's case may also have originated from a ciliary nerve, but it was so far back that it did not grow within the bulb.

It is my belief that a number of the leukosarcomas which have been described in the past belong in the group of neurofibromas. As an

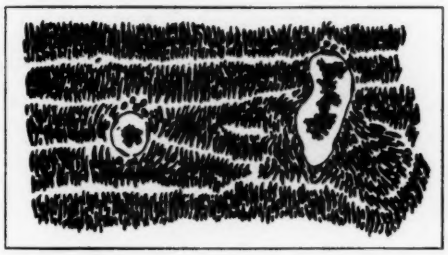

Fig. 3.—Slender spindle-shaped tumor cells, which are arranged in parallel rows. All the spindles are free from pigment; in this illustration they are shown dark because the author (Schieck) was interested less in the cells as such than in their arrangement in rows (from Schieck,[11] fig. 18).

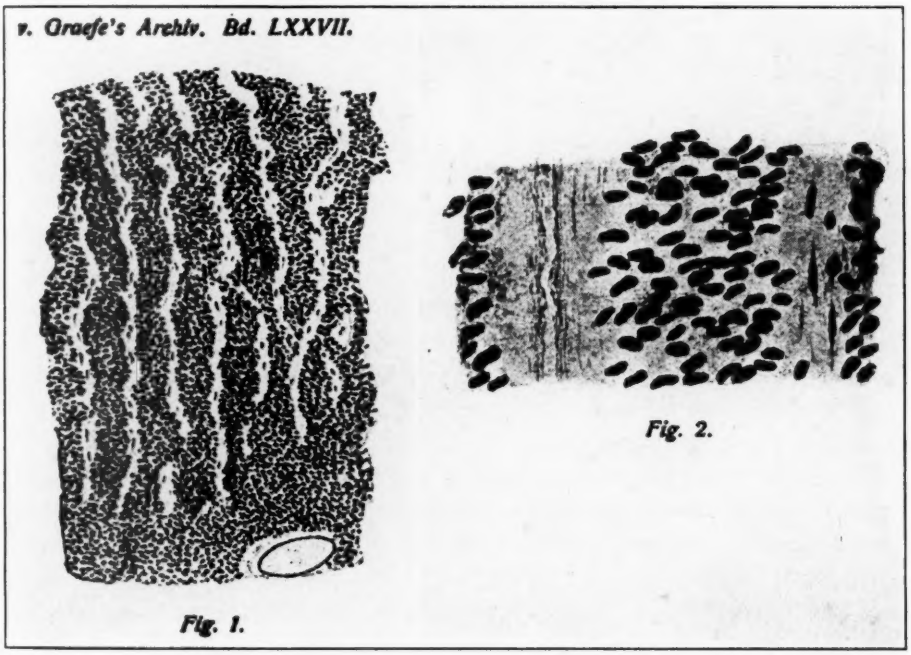

Fig. 4.—Reproduction of figures 1 and 2 of Fuchs.[12]

example, in his book on melanosarcoma of the uveal tract F. Schieck [11] described a tumor (illustrated by his figure 18) similar to this one

11. Schieck, F.: Melanosarcoma of the Uveal Tract, Wiesbaden, J. F. Bergmann, 1906.

except that there were chromatophores and pigmented spindle cells in the outer layers. However, these pigment-bearing cells could well have been normal choroidal pigment cells caught up in the tumor.

E. Fuchs [12] reported a similar case (fig. 4). His description, in part, is as follows:

> The nuclei are in long bands lying parallel to one another, with their long axis vertical to the axis of the bands. The nuclei are imbedded in a homogeneous interstitial substance in which no cell borders are distinguishable so that perhaps there is a kind of syncytium. The nuclei are not lying bare to one another but in irregular rows behind one another; on the number of these rows, varying from 2 to 10, depends the breadth of the nuclear bands. The interstitial space between two bands is filled with a homogeneous substance which is usually vacuolated (frequently such is found between the nuclei in the bands and so appear to be an artefact developing through hardening). A clear framework between the bands is not seen. In one case in the interstitial substance between the nuclear bands are single round nuclei; in another case single long nuclei the axis of which is vertical to those of the bands and are perhaps connective tissue. Occasionally between the bands are single pigment granules, though usually the tumor is not pigmented. The sparse blood vessels are not directed towards the nuclear bands; they cut crosswise to them very frequently. I have observed this structure in three cases, in one case in the entire tumor, in two others in single sections.

I believe that with the special stains of today these tumors would be found to have originated from nervous tissue.

SUMMARY

A case of intra-ocular neurofibroma or perineurial fibroblastoma has been described. The outstanding characteristics were the regular arrangement or palisading of the cells, the presence of reticulin fibers, cystic changes and the absence of pigment and of mitotic figures.

A review of other intra-ocular neurofibromas as compared to this one is given, showing that some tumors described in the past under the title of melanosarcoma and leukosarcoma may well belong in this group.

A discussion of the neurofibromas of the orbit has purposely been excluded in this presentation, though it is readily seen that such a tumor as I have described arising from a ciliary nerve may be the intra-ocular expression of changes which could also affect only the orbital portion.

12. Fuchs, E.: Sarcoma of the Choroid, with Remarks Concerning Necrosis of the Uvea, Arch. f. Ophth. **77**:304, 1910.

SURGICAL MANAGEMENT OF PTOSIS

WITH SPECIAL REFERENCE TO USE OF THE SUPERIOR RECTUS MUSCLE

P. CHALMERS JAMESON, M.D.

BROOKLYN

It is my purpose in this paper to express my understanding of the surgical management of ptosis, for in spite of the fact that so much has been written about it there is still a diversity of opinion on the subject. Many differing valuations have been placed on operations for ptosis, which in the aggregate possibly outnumber any other ophthalmic procedure. Wilder,[1] who has written one of the best articles descriptive of the condition, enumerated and described no less than forty-three operations and modifications. Such multiplicity of procedures means that no one operation has been adapted to all types of cases.

It almost goes without saying that an adequate survey of all the conditions present should be made and a computation arrived at as to how much intervention should be done and the best manner of doing it. This survey should differentiate between the varying forms, for instance, the congenital paralytic, the partial paralytic and the non-paralytic, the obstructive, the integumental and the trachomatous and symblepharon types and also conditions which impart much bulk and burden to the lid.

Paralysis of a muscle of the globe may contribute to ptosis. Advancement of a semiparalytic superior rectus muscle supplemented with advancement of the inferior oblique muscle has been known to cure traumatic ptosis without operation on the lid. Paralysis of the inferior rectus muscle may give the impression that the lid is at fault. There is a rare form of ptosis accompanied with extreme ptosis of the eyebrow probably associated with paralysis of the occipitofrontalis muscle.

I shall first advance certain measurements and relationships which I have not seen reported as associated with ptosis in the ophthalmic literature but which contribute materially when making one's computation.

Read at the Seventy-Third Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 4, 1937.

1. Wilder, W. H., in Wood, C. A.: System of Ophthalmic Operations, Chicago, Cleveland Press, 1911, vol. 2, p. 1522.

First I shall mention one of much importance, i. e., the relation of the margin of the upper lid to the globe. The margin of the upper lid is always to be found in the normal person on a line midway between the pupillary margin and the upper limbus. This relation is scrupulously maintained in the primary position, i. e., when the eyes are looking straight forward, and for the most part when the gaze is directed up or down, the levator muscle of the lid and that of the eyeball acting in great harmony. It is one of the striking phenomena of perfect ocular adjustment. If the margin of the lid drops below this line, ptosis in some form is likely to exist, and the drop registered in millimeters is the amount of the ptosis. For instance, if the margin of the upper lid should bisect the pupil, the approximate ptosis would be about 3.5 mm.

Second in order of mention is the measurement of the palpebral opening, both vertical and horizontal. The average normal vertical measurement is some 9 mm.; anything less, with elimination of a possible pathologic condition, would point to ptosis.

Third to be considered is the horizontal measurement of the palpebral opening. The average horizontal measurement of the palpebral opening is 27 mm. Measurement of this dimension should not be omitted, as it throws light on the question as to whether canthotomy should be performed to aid in the uplift of the lid. This is valuable especially if epicanthus exists. In this condition the abbreviation of the lid may obstruct the uplift.

Fourth, the distance between the margin of the lid and the arch of the eyebrow should be measured with the eyes in the primary position. In the normal young subject this measurement varies from 15 to 18 mm., but in a subject with congenital ptosis it may run up to 25 or even 30 mm. This measurement is valuable to computation as helping in the estimation of the effort of the occipitofrontalis muscle. It is therefore a guide to gradation. It is the stretching of this space that imparts to persons with ptosis the distraught appearance so noticeable.

The behavior of ptosis in the upward and the downward gaze should also be noted, and, as brought out later in this paper, the vertical measurement of the superior tarsus from the top of the arch to the margin of the lid should be taken. This measurement is almost constant at 8 mm. in the adult.

These measurements undoubtedly contribute more accurately to the ophthalmologist's knowledge of the case and guide him in selecting the operation to be performed and the amount of gradation to be undertaken. They convey these essentials to him much more accurately than would a cursory glance.

DIVERSE OPERATIVE PROCEDURES

Ptosis lends itself to many diverse operations, and it is a wise precaution for the operator to select the procedure best adapted to correct. It is hardly possible for one to describe all these procedures, and I can only briefly comment on some of them, most of which I have at some time tried and practiced.

Cleancut oval excisions, when redundant integument exists or to supplement various operations, are valuable. They heal well without evidence if the subcutaneous suture is used.

The flap-tucking, transplanting cutaneous operations, on the other hand, are often attended by ugly puckering. An exception to this may be the Hess operation.

Operations for tarsal resection, for example that of Gillet de Grandmont, are excellent in cases of moderate ptosis in which the operator does not have to deal with excessive weight or bulk. The result is sometimes excellent in cases in which there is still some action of the levator muscle. It stands to reason that shortening of the tarsus will, in a measure, raise the plane of the margin of the lid. Suture operations, and by these I refer to the types of operation in which the operator relies on the irritation produced by the transit channel to contract and hold the drooping tissues, are often disappointing and are usually not attended by much correction. Eversbusch [2] stated that the advocates of these procedures in his day looked on infection in the line of suture as desirable.

Among the operations best calculated to correct the heavy lid is the Fergus operation, i. e., the direct transplantation of a tongue of the occipitofrontalis muscle into a pocket between the tarsus and the orbicularis muscle. The operations consisting of incision of the tarso-orbital fascia and advancement of the levator muscle to the margin of the lid are not unattended with danger, as they open into the post-orbital region, and if the incision in the tarso-orbital fascia is of any extent a troublesome hernia of areolar fat may follow. I have abandoned this procedure, as in the last case in which I used it I had difficulty in pressing back all the areolar fat which presented.

A much better procedure and another of the operations on the heavy lid is the operation of Eversbusch. This amounts to folding the tarso-orbital fascia and adjacent tissues, including the levator muscle, into a pocket on the surface of the tarsus. Not only is this operation a corrective procedure which can be graded, but it furnishes a certain amount of curvature to a flattened lid such as frequently exists in extreme congenital ptosis. In my experience, the Eversbusch operation is one of the best, if not the best, of its type.

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2. Eversbusch, O.: Zur Operation der congenitalen Blepharoptosis, Klin. Monatsbl. f. Augenh. **21**:100, 1883.

It is noticeable that while these types of operation do not furnish elevation of the lid by the eyeball as the operation on the superior rectus muscle does, they do elevate the lid through the instrumentality of the occipitofrontalis muscle and in many cases give a more even, pleasing and normal picture to the lid than the operation of the superior rectus muscle, which at times gives the appearance of an exaggerated central arching of the margin of the lid.

Another operation on the heavy lid is the Hess operation, which has already been mentioned. In this procedure, when it is associated with the Beard modification, in addition to the tucking of the skin from the under surface, the tarso-orbital fascia is picked up in transit and approximated to the occipitofrontalis muscle. This is a stronger procedure. It amounts to folding of the levator muscle and fascia in the reverse direction to that in the Eversbusch operation.

These operations for shortening the tarso-orbital fascia in which margin of the lid is approximated to the occipito-frontalis muscle by bringing the tarso-orbital fascia down to the margin, as in the Eversbusch operation, or by bringing the lower part of the fascia up to the occipitofrontalis muscle are not only valuable in cases of much heaviness of the lid but, of course, impart a material quality of intermittent natural motion through the instrumentality of the occipitofrontalis muscle. I say natural because the motion is frequently used when no ptosis exists, for instance, in conditions of astonishment, fear, horror, surprise or consternation.

Motais [3] was the first to practice the operation which gave mobility to the lid through the instrumentality of the superior rectus muscle. It was a brilliant conception of a great anatomist. His first operation was performed on April 20, 1897. In his original article, published in the *Annals d'oculistique,* he stated that he was drawn to the procedure by several observations, anatomic and physiologic, among which were the common origin and the common direction of the superior rectus muscle and the levator muscle, their common aponeurotic envelopment, their common source of innervation and their common physiologic concord, both acting in perfect unison together. He stated: "From these facts we must conclude the two elevators are admirably disposed to augment each other reciprocally." He added, commenting on conditions subsequent to the operation, "But above all what is demonstrated by copious evidence very well founded on anatomic and physiologic considerations that have guided us is the perfect manner with which the eyelid is raised and folded in the upward glance." It is a tribute to this great ophthalmologist that most present day ophthalmologists have witnessed these conditions, at least when the operation is not made to exceed its limitations.

3. Motais, E.: Ann. d'ocul. **118**:5, 1897.

From his writings one would judge Motais felt that he obtained independent action from the levator muscle after the operation. This, however, is not the case.

Shoemaker [4] published a paper in 1907 which established the fact that after a Motais operation was performed no independent action was imparted to the levator muscle and that any motion which the lid obtained was purely through the movement of the eyeball which elevated or depressed it. After reading this paper a personal analysis must persuade one that the Motais operation induces a virtual symblepharon between the lid and the eyeball.

At first glance this lack of independent gliding may not seem a detriment, especially when one recalls the fact that the upper lid constantly retains its one position in relation to the eyeball in the upward and the downward gaze, but, as will be seen later, it has an important bearing when the eyeball and the superior rectus muscle are overloaded with a heavy lid.

It might be well to review briefly the main points of the technic of Motais: the isolation of the muscle (the superior rectus muscle), the dissection of a tongue of muscle 3 mm. wide and 10 mm. long, the incision through the tarso-orbital fascia and the levator muscle through which the tongue of muscle is passed and drawn into a pocket between the orbicularis muscle and the tarsal surface and, finally, the anchorage of the tongue 2 mm. below the curve of the tarsus.

Shoemaker modified the Motais procedure by combining the prepared dissection as in Motais' operation with the adjustment of the lid by exposing the field on the outer surface, and recently Kirby [5] advocated an approach altogther from the outside, the incision being made directly down to the muscle.

One cannot help asking the question why, if the end of the tongue in the Motais operation was to be fastened 2 mm. below the curve of the tarsus, was it necessary to make a dissection 10 mm. long when the tongue was extended only some 3.5 to 4 mm.? It could not well be for reasons of anatomic adjustment, as, according to the law of lateral incisions whereby one lip shortens and the other lengthens, the adjustment should be obtained as readily with a 4 mm. dissection as with a 10 mm. dissection, and less mutilation would have been necessary. Why did Motais not attempt to grade his operation when necessary by bringing the end of the tongue farther down on the tarsus, to the margin of the lid, as is often done in these days, and why was it necessary to mutilate the muscle at all by making the dissection of the tongue when the full strength of the unmutilated muscle could be used?

4. Shoemaker, W. T.: Ann. Ophth. **16**:608, 1907.
5. Kirby, D. B.: Arch. Ophth. **57**:327 (July) 1928.

Incidentally, with the Motais operation it is as safe to make a tongue of two thirds of the central portion of the muscle, making it 6 mm. broad instead of 3 mm. The outer two sixths left on the outside prevents retraction, and anchorage to the tarsus holds the central portion of the muscle. If this is done it is in a measure preventive of the accident whereby the attenuated tongue parts at the junction of the muscle and the tendon, and in addition constitutes a much stronger operation.

It was Parinaud [6] who first attacked Motais' operation on the score of unnecessary dissection. He stated that he had had Motais' idea in mind but did not put it into practice. He wrote:

> The idea of a like operation was suggested to me several years ago by an intern who attended my clinic, M. Deroque, who cited to me the analogous procedure practiced in general surgery. I judged then that the operation was unpractical and not suitable to give sufficient elevation of the lid. I was mistaken, since M. Montais had accomplished it, but the objection which I made still persists.

Parinaud's article on his operation appeared in July 1897, just after Motais' paper in May. The procedure had for its object the direct attachment of the unmutilated muscle to the arch of the tarsus, with no dissection of the tongue. There is no doubt that the Motais operation succeeds in many cases, although it is a failure in others. There is no doubt in my mind, however, that operations based on the Parinaud conception are much the stronger operations, and the Motais operation accomplishes nothing which the Parinaud procedure cannot excel and by giving the muscle stronger purchase insures a larger return of correction.

If, as Shoemaker has shown (and he undoubtedly has), a virtual symblepharon between the tarsal curve, the muscle and eyeball is established with the Motais operation and there is therefore no independent action of the lid, why not establish that agglutination at a point where it will be most efficacious, leaving the muscle unmutilated and shortened and in the strongest condition to maintain the added burden? The conception of the Parinaud operation is the primary raising and fixing of the lid to a permanent position on the eyeball, the muscle being left unmutilated and strong so that in the upward gaze the eyeball will propel the superimposed lid with it. In both Motais' and Parinaud's original article there seems to be a singular absence of consideration of what I believe to be a most important question, i. e., gradation and shortening of the muscle. Motais, as has been stated, could have obtained 5 mm. more shortening of the central portion of his muscle if the tongue had been advanced to the margin of the lid,

6. Parinaud, H.: Ann. d'ocul. **118**:13, 1897.

but he left it fastened only 2 mm. below the tarsal curve. Fastening of the end of the tongue to the margin of the lid is frequently practiced today.

Parinaud, on the other hand, did grade his operation. In one brief statement he dealt with this phase of the subject. He stated: "Increase in effect can be obtained by placing the sutures farther back on the muscle." This sentence, when analyzed, embodies the idea of grading and shortening the muscle. Wilder made the statement that it is often claimed that the weak superior rectus muscle acquires strength under the additional work thrust on it. I am confident that it is the shortening of the muscle that is responsible for the development of the strength and not the superimposed burden, and just as one shortens any other muscle to impart strength, so one should shorten the superior rectus muscle to strengthen it to carry its added burden. By doing this one will obtain a higher correction.

The taking of a weak attenuated muscle hardly capable of performing its own function and placing on it the responsibility of carrying probably two or three times the normal weight is the most prominent cause of failure of operation on the superior rectus muscle.

It may be reasoned that shortening to any extent prevents closure of the lid, especially during sleep. The mother shortly after the operation may state that the child sleeps with the eyes open. I have always found that the orbicularis muscle soon trains itself to bring about closure and that this symptom disappears quickly. A Buller shield is a good dressing in the interim.

If there is any excuse for adding another operation for ptosis to the already swollen number it is because of this important preparation by gradation and shortening and also some other features which make for increased effect and security. In presenting this operation I should first pay tribute to Professor Motais for his pioneer conception of the use of the superior rectus muscle, to Profressor Parinaud for what I believe to be a stronger concept of its use, and to others, especially Dr. Shoemaker, for valuable observations.

REPORT OF A NEW OPERATION

In the device which I report for the first time and which I have practiced in eight cases with success I have adopted the following principles: 1. The principle of the use of the entire strength of the unmutilated superior rectus muscle. 2. A direct incision into the cartilage, with entrance into a pocket prepared for the reception of the folded muscle, instead of the incision into the levator muscle or the tarso-orbital fascia into the postorbital region. 3. Shortening of the muscle and consequent elevation of the lid by the folding of the attached

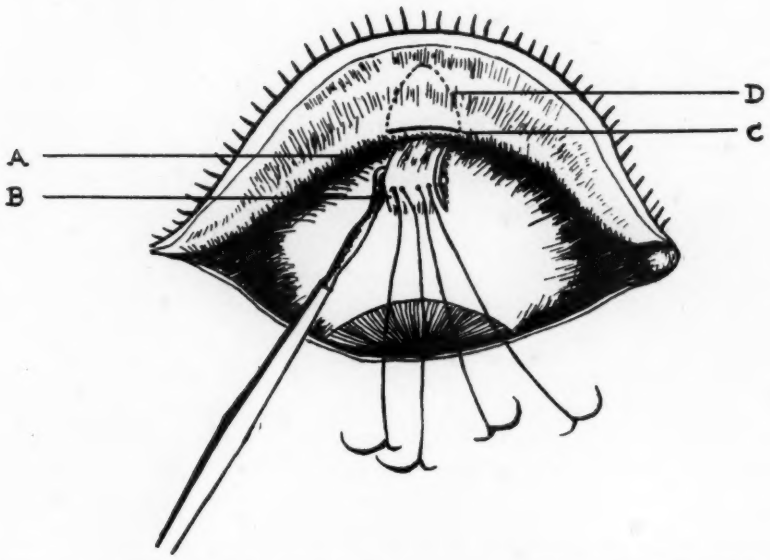

Fig. 1.—After the lid is everted the superior rectus muscle is dissected out but not detached (*A*). Two double-armed sutures are introduced from under the surface; the farther back on the muscle these are introduced, the greater the shortening (*B*). An incision is made directly into the cartilage about 2 mm. below the upper surface of the tarsus (*C*). A pocket is made between the orbicularis muscle and the tarsal surface (*D*).

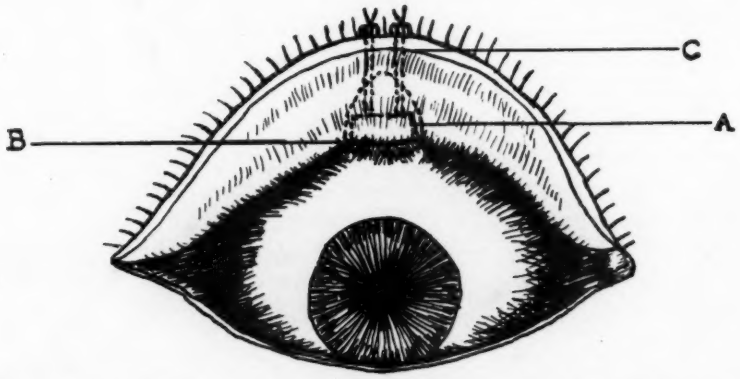

Fig. 2.—The superior rectus muscle is folded on itself by being drawn into the pocket (*A*). Traction is made until the insertion of the muscle approximates the primary incision of the cartilage (*B*). Sutures are passed between the surface of the tarsus and the orbicularis muscle and are tied at or near the intermarginal space (*C*).

muscle on itself prior to its introduction into the pocket and an added method of elevating the tarsus by placing the incision in the cartilage farther below the curve. 4. The secure attachment of a folded shortened muscle.

The technic is as follows, the approach being accomplished altogether from the conjunctival side:

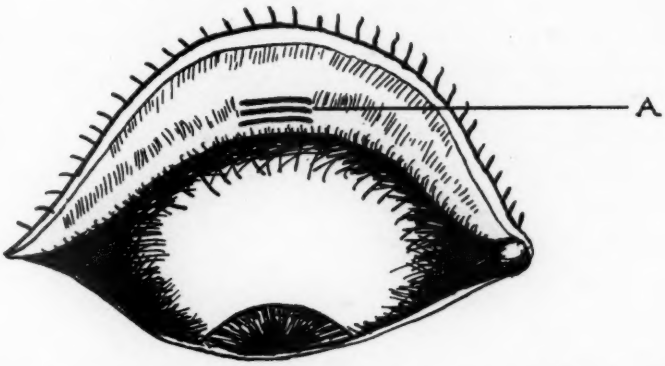

Fig. 3.—Increased gradation can be obtained by: placing the aperture of the pocket lower down on tarsus (*A*), introducing the two double-armed sutures farther back on the superior rectus muscle (fig. 1 *B*) and approximating the end of the muscle (by traction) nearer the intermarginal space (fig. 2 *B*).

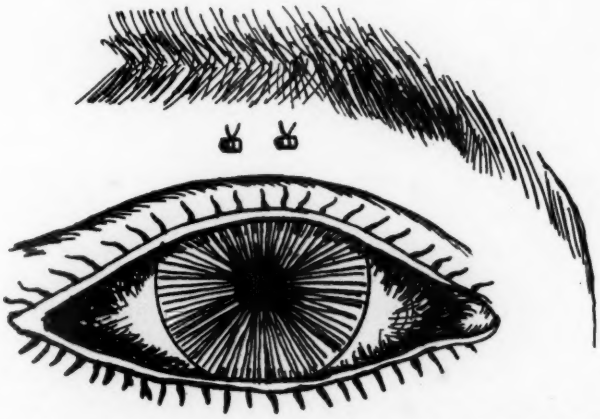

Fig. 4.—Mattress sutures may be introduced underneath the lid through the cartilage and the muscle and tied over bolsters on the outer surface of the skin if more secure anchorage is desired (*A*).

1. The superior rectus muscle is dissected and exposed in the usual manner, the muscle being free to admit of as much shortening as desired (fig. 1, *A*).

2. Two double-armed sutures are introduced and are looped underneath the muscle as far back as shortening is desired. Making use of this gradation is important (fig. 1, *B*).

3. An incision is made directly into the cartilage (not into the levator muscle), large enough to admit the muscle into a pocket prepared for it (preferably 2 mm. below tarsal curve, and lower if greater elevation of the lid is desired) (fig. 1, *C*).

4. The muscle is overlapped on itself by drawing the sutures and the muscle into the pocket prepared for it (fig. 2 *A*).

5. The sutures and the muscle are secured in the pocket by bringing the running sutures through to the margin of the lid and tying them on bolsters (fig. 2, *C*).

6. Increased gradation to the extent of 4 mm. can be accomplished by bringing the aperture of the pocket lower down on the tarsus (fig. 3, *A*).

7. Gradation can also be attained by approximating the end of the muscle (by traction) nearer the intermarginal space (fig. 2, *B*).

Finally, the mattress sutures are brought through the tarsus, the superior rectus muscle, the orbicularis muscle and the skin, and the suture is tied on the surface of the skin as supplementary anchorage (fig. 4).

COMMENT

I do not present this operation as a substitute for each and every other method, believing, as the theme of this paper would indicate, that the ophthalmologist has a wide field to select from and can well choose the operation or combination of operations best adapted for his needs.

This new operation as I have presented it is, as are all operations on the superior rectus muscle, appropriate for ptosis with a moderate superimposed burden of tissue. When the bulk of the lid is excessive, one of the operations performed when there is a heavy burden on the muscle may well be substituted or used as a supplement.

If the Motais technic were practiced it could be made a stronger procedure (1) by using two thirds of the breadth of the muscle, and this with equal safety; (2) by lessening the 10 mm. length of the dissection, and (3) in contrast to the original Motais procedure, by making the incision, as in my own operation, directly into the cartilage instead of through the levator muscle at its junction with the tarsal arch.

I believe, however, that the new operation as here presented is the best operation on the superior rectus muscle at the command of the ophthalmologist. In my judgment, it is a stronger, simpler, quicker and less complicated procedure than the Motais operation and one which by reason of its flexibility of gradation and shortening is attended with a higher return of correction.

SUMMARY

In review the following may be stated:

1. In every case of ptosis the relations of the margin of the upper lid to the globe, measurement of the vertical and horizontal dimensions of the palpebral opening and measurement of the distance between the margin of the lid and the arch of the eyebrow with the eyes in the primary position as described should be taken, and the selection of operation should be based on as accurate a computation as possible.

2. Frequently no single operation will best correct the condition in every case, and combinations making for the most perfect correction may often be used.

3. Operations on the superior rectus muscle frequently fail, owing to the incapacity of the muscle to bear the increased burden.

4. Gradation not only is useful to correct ptosis but is necessary and can be accomplished by four methods, as in my operation: (*a*) introducing sutures at varying distances from the insertion of the muscle (the farther back these are introduced the greater the gradation) (fig. 3, *B*); (*b*) folding the muscle on itself in drawing it into the pocket (fig. 2, *A*); (*c*) lowering the aperture of the pocket on the tarsus (fig. 3, *A*); (*d*) approximating the end of the muscle by traction nearer the intermarginal space (fig. 2, *B*).

5. Utilization of the unmutilated muscle makes for a stronger procedure than the method in which a tongue is used.

6. An incision directly into the cartilage for the entrance of the prepared pocket is preferable to an incision into the postorbital region through the levator muscle and the tarso-orbital fascia.

FORMATION OF PRERETINAL CONNECTIVE TISSUE IN THE VITREOUS IN ACUTE CHOROIDITIS

REPORT OF THREE CASES

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The opacities of the vitreous which accompany acute exudative choroiditis usually become absorbed in the course of time and do not interfere with vision unless the choroidal focus invades the macular region. Sometimes a band of opacity remains which extends from the nerve head to the area of choroidal atrophy. Another and more unusual permanent change is the opacity of the vitreous which is the subject of this communication. The vitreous is clear except for this opacity, which is situated just in front of the retina and seems to have a predilection for the macular region. The opacity adheres to the retina and is irregularly branching, whitish, and of varying thickness in the center, fading out at the periphery. Stationary, it seems anchored to the internal membrane of the retina. It always covers the retinal blood vessels, and new-formed capillaries are never observed to penetrate the opacity. In this respect it differs from retinitis proliferans. Nor does it ever detach the retina or even exert any traction. The opacity tends to become thinner, though it never disappears, and final vision is often surprisingly good. One characteristic is that the choroidal lesion is always a large one and is not connected with the opacity of the vitreous. This condition has been observed in the three following cases:

REPORT OF CASES

CASE 1.—V. R., a man aged 22, was seen on March 20, 1935. He gave the history that the sight of the left eye became affected on January 3. The vision in the left eye was 20/200. There were many posterior corneal deposits, membranous cobweb opacities in the vitreous and a large area of choroidal exudate in the nasal periphery. The tuberculin test was positive, and he was given tuberculin and the usual general and local treatment. The clearing of the vitreous took place very slowly. On November 23 the vision was 20/100, and the choroidal area had healed. The opacities in the vitreous gradually cleared except for the large stationary formation in the macular region, which was preretinal, irregular and branching and definitely anterior to the blood vessels. Vision slowly improved, and the macular figure cleared to an unusual extent. Final vision was 20/30.

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CASE 2.—J. F., a man aged 39 years, was seen on Feb. 1, 1937. He stated that his right eye had become affected eight years before. He received no treatment and could give little information about his case. Vision with a glass was 20/200. The vitreous was clear. There was a preretinal organized opacity in the vitreous in the macular region, which was not connected with the large atrophic choroidal scar, extending downward and outward. The opacity in the vitreous was fixed, anterior to the retinal vessels and had the same irregular, branching structure noted in the other cases. (This condition is illustrated in the figure.)

Preretinal organized vitreous opacity.

CASE 3.—C. S., a youth aged 17, was seen on Oct. 15, 1935. He said that the sight in the right eye became affected in June. On examination vision in the right eye was 8/200. There were many membranous opacities of the vitreous; a stationary opacity was situated in front of the macular region, and another partly covered the disk and extended down in front of the inferior retinal vessels. A large active choroidal lesion was present in the lower and outer periphery. As the patient reacted to tuberculin, he was put on a regimen of treatment with tuberculin, sunlight baths, rest and tonics. On Jan. 7, 1936, vision had improved to 20/50—. The vitreous changes had diminished and now consisted of a branching opacity extending across the optic disk and adherent to the retina in places. The choroidal focus was healing. After spending the rest of the winter

on a ranch in Arizona, with continuation of treatment with tuberculin, he had vision of 20/20 — on April 6. The opacity of the vitreous had largely cleared, and the choroidal area had healed. A curvilinear opacity was noted in the vitreous, with broad, branching extensions adherent to the retina. There was a connection with the optic disk and some of the vessels just below, though no vessels could be seen entering the opacity, and in no place was there a suggestion of a detachment of, or even traction on, the retina.

COMMENT AND SUMMARY

This clinical picture consists of a large choroidal patch with the corneal deposits and vitreous opacities usually present in cases of acute choroiditis. As the process heals the vitreous clears except for a curious fixed opacity which is adherent to the retina, particularly in the macular region, and is not connected with the area of choroidal atrophy. The opacity becomes very faint in places, and surprisingly good vision is sometimes obtained. No new-formed vessels pass into the opacity, and there is no suggestion of detachment of or traction on the retina.

PATHOGENESIS OF DISCIFORM DEGENERATION OF THE MACULA

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In 1875 Pagenstecher gave a description of the macroscopic and microscopic appearance of a macular lesion observed in an eye after its removal for adherent leukoma and secondary glaucoma. His descriptions and illustrations of this lesion leave little doubt that the eye was affected with what is now known as disciform degeneration of the macula. His is the first case of this disease that we were able to find recorded in the literature. He designated the condition chorioidioretinitis in regione maculae luteae. The first employment of the term degeneratio maculae luteae disciformis was by Oeller in 1905. In 1926 Junius and Kuhnt adopted this term, substituting, however, *Netzhautmitte* for macula lutea, and definitely established the condition as a disease entity. Other terms to designate the disease, employed prior to the publication of their monograph, are indicated by the titles in the appended bibliography. The term favored by Junius and Kuhnt, although in several respects misleading, has now been generally accepted. Following is an analysis of all the cases of senile disciform degeneration at the macula reported in the available literature. There were eighty-four such cases, one hundred and twenty-nine eyes being affected. Thirteen of these eyes were examined microscopically. The three cases to be described in this communication are not included in the analysis.

ANALYSIS OF RECORDED CASES OF SENILE DISCIFORM DEGENERATION OF THE MACULA

Race.—No patient was stated to be other than white, but only a few reports specified the race.

Frequency.—There is no doubt that the cases described in the literature represent only a fraction of the total number of cases recognized as instances of disciform degeneration at the macula. Kahler and

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Read at the Seventy-Third Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 5, 1937.

O'Brien stated that twelve cases were observed in the ophthalmologic clinic of the State University of Iowa in one year.

Age.—In the majority of cases the condition occurred in the fifth and sixth decades of life. The youngest patient was aged 39 (Neame); the oldest was 83 (Kahler and O'Brien). The average age for the entire group was 68. Age, therefore, was a definite factor.

Sex.—Of the eighty-four patients, forty-seven were male and thirty-four female; the sex of three was not stated.

Trauma and Inflammation.—There was no evidence that these were factors in the disease.

General Condition.—Many of the reports contained little information other than a description of the ocular lesion. Nineteen of the patients were reported to have general hypertension. Fifteen had general arteriosclerosis. Eight had a cardiac condition, and four were nephritic. Four were diabetic. Only three were syphilitic. One patient was known to have lived eleven years after the ocular condition was first observed. Eight patients died of coronary disease.

Eye Affected.—In thirty-nine cases the condition was unilateral, the right eye being affected in eighteen. Among twenty-one of the cases in which the condition was bilateral it was determined that the right eye was the first affected in twelve.

Symptoms and Mode of Onset.—Central vision in the affected eye was in all cases greatly impaired or completely abolished when the patient first came under observation. Some patients noted a positive central scotoma. No important improvement in vision occurred in the progress of the disease in any case. As to whether the disease had the same mode of onset in all cases, and, if so, whether this was gradual or sudden, it was impossible to determine. The fact that only gradual loss of vision was noted by most patients may have been due to preceding development of senile macular changes which prevented any subjective recognition of the onset of disciform degeneration, even if this onset was actually abrupt. In a few cases the patient noted disturbances in vision such as loss of visual acuity, metamorphopsia and photopsia before any definite lesion could be seen with the ophthalmoscope, but here again it is not clear that these were prodromal symptoms of disciform degeneration. On the other hand, when a patient stated that his loss of vision was sudden, it may actually have been gradual and only suddenly discovered. The only positive evidence regarding the mode of onset was that furnished by twelve eyes. In each of these the onset was undoubtedly sudden, since a subretinal hemorrhage was observed at the first ophthalmoscopic examination.

Aside from Pagenstecher's case, in which there was glaucoma secondary to an adherent leukoma, glaucoma was not known to have developed in any case.

Ophthalmoscopic Signs.—In twelve eyes there was first observed a subretinal hemorrhage in the macular region, which elevated the retina in the form of a mound. Adjacent to this were other hemorrhages. These, which were usually described also as subretinal, often outlined the ultimate mass. More remote hemorrhages were apt to be described as occurring within the retina. These were observed to disappear, and new ones were seen to occur in other places. In cases, comprising the majority, in which the lesions were first observed at a later stage, hemorrhages were less frequently noted. However, in seventy-five of the one hundred and twenty-nine eyes hemorrhages were noted, and in thirty-six eyes hemorrhages adjacent to the mound were still in evidence.

The mound as seen early was described as grayish, greenish or almost black. Later it became white except for more or less deposition of pigment. The retinal vessels always were visible in front of the mass. In several cases arteriovenous communications were noted. The size of the mound varied from one-half to several times the size of the optic disk. In some cases its height was considerable, in one case reaching 6 D. In some cases it was dimpled over the hypothetic macula. It was usually more elevated early in the progress of the disease and often appeared to "settle" as the disease progressed.

The margins of the mound were usually distinct. Almost always at least a portion of the edge was sharply etched against the adjacent retina. The shape was often disciform but sometimes assumed other contours. The location of the mass was the macular region between the superior and the inferior temporal vessels. Rarely did the lesion exceed the limits of these vessels.

Associated Fundic Changes.—Sclerosis of the retinal vessels was recorded as existing in only twenty-five out of the one hundred and twenty-nine affected eyes. In each of the five cases in which the condition later became bilateral the second eye showed senile macular changes before the development of the disciform lesion. These senile changes included drusen, pigmentary changes, and white spots in the papillomacular region and along the macular vessels. Among thirty-seven cases in which the condition was unilateral the fellow eye showed senile macular changes in fifteen and was reported normal in only sixteen (four eyes had senile cataract, which prevented examination of the fundi, and two had been enucleated for other causes).

In sixty-one eyes some type of white or yellowish white spots of degenerative or exudative character were found near the lesion at some stage of its progress. These ranged from small spots the size of a pinpoint deep in the retina to frank circinate retinitis (seen in twenty-two eyes). In two of the cases of the latter condition, circinate retinitis was observed first; in one of these two, disciform degeneration was found in addition fourteen months later, and in the other, a few months

later. In the other cases circinate and disciform lesions were seen simultaneously, and it was impossible to determine which condition preceded the other. In a case (reported by Spicer in his discussion of Lawford's paper) not included in this series, circinate retinitis existed in the fellow eye, and we have recently seen another such case. In the description of one case, which was briefly reported by Neame, it was stated that angioid streaks were present in the other eye. In a case in which the condition was unilateral, reported by Davis and Sheppard, which will be referred to later, angioid streaks were present in each eye. Feingold stated that "similar changes in the macula were observed in a patient showing all characteristic fundus changes of angioid streaks," but gave no further details regarding the eye.

Microscopic Observations.—Thirteen eyes were examined microscopically. One eye was removed for glaucoma that had occurred secondary to an adherent leukoma; four were removed for suspected tumor, and eight were removed post mortem. The observations can be briefly summarized as follows:

1. The retina over the mass was observed to show degenerative and atrophic changes in all but two cases. These were believed by most observers to be secondary changes.

2. The pigment epithelium in all the cases was seen to be markedly proliferated and was a prominent component of the mound.

3. In six of the eyes examined microscopically the lamina vitrea was observed to show pathologic changes. Michel, Verhoeff and Behr noted colloid excrescences on it, as well as variability in its staining properties and thickness. Six eyes were seen to show breaks in Bruch's membrane through which blood vessels, red blood cells or lymphocytes passed. In four instances this membrane was reported to be intact and normal.

Other than showing signs of compression, the choroid of eight eyes was observed to be completely normal. Elschnig noted atheromatous choroidal changes. Verhoeff reported the choroidal vessels to be slightly sclerotic. Hanssen noted small collections of lymphocytes in the choriocapillaris in the region of the lesion, and Behr observed proliferation of the intima of the middle-sized arteries.

The mound was uniformly reported to consist of proliferated pigment cells and chiefly of fibrous tissue. Behr reported strands of elastic tissue, which he believed lifted off from the outer fourth of the lamina vitrea. Small amounts of blood and albuminous exudates in various stages of organization were occasionally observed. Vessels presumably chiefly from the choroid were observed within the mound in all cases, and occasionally a capillary was observed to enter it from the retina. Choroidal vessels were seen by several observers to penetrate the lamina

vitrea. Michel noted cartilage within the pseudotumor, and Axenfeld reported an area of ossification within the mass.

Pathogenesis.—Elschnig placed the origin of this disturbance in the retina and its vessels, and Junius and Kuhnt, in the inner layers of the retina; Ormond, Duynstee and others believed that the disease originated from the capillaries of the outer retinal layers.

Michel contended that the origin of the growth was in the lamina vitrea. Behr evolved a theory of vital functions which he attributed to the lamina vitrea and contended that a pathologic disturbance of this membrane interferes with these functions and stimulates hyperplasia.

Hanssen, who noted lymphocytic infiltration of the choriocapillaris, concluded that the choroid was the chief offender.

Pagenstecher, Axenfeld, Hegner, Neame, and Coppez and Danis subscribed to an inflammatory cause.

Oeller, Batten, Beatson and Knapp believed that the mass originated from a subretinal exudate or hemorrhage or both.

Possek thought that the exudate was caused by arteriosclerotic changes in the tissues of the eye. Sclerotic changes in the choroidal vessels particularly were observed and emphasized as a possible cause of nutritional disturbance with resultant hyperplasia by Cords, Pallarés, Verhoeff and Paul.

JUVENILE DISCIFORM DEGENERATION OF THE MACULA

In the literature there have been described cases in which a macular condition resembling senile disciform degeneration has occurred in persons below the age of 40 years. For convenience we shall refer to it as juvenile disciform degeneration of the macula. It differs from the senile type also in the fact that the lesion tends to heal, leaving few, if any, changes and slight or no impairment of vision. Just how frequently it occurs is problematic, because it may often have been mistaken for some other condition. Special attention was called to this condition by Junius in 1929. He reported four cases under the classification juvenile retinitis exsudativa macularis, three of which seem to us to belong to the type under consideration. The ages of the patients were 34, 32 and 27 years, respectively, and only one eye was affected in each case. In the first case the condition reached its height in ten weeks, and vision was then reduced to counting of fingers at 2 meters. In six months the lesion had entirely disappeared and vision was normal. The patient was seen again twenty-four years later, at which time the fundus and vision were still normal. In the second case there were two small hemorrhages at the margin of the lesion. Vision was reduced to perception of movements of the hand. At the end of two months the lesion had largely disappeared, leaving a pale oval area partially demarcated by a fine pigmented line, and the vision was 0.3. In the other case, the

onset was sudden, and vision was reduced to the counting of fingers at 0.75 meter. Whether vision later improved was not determined, because the patient was lost sight of. The patient was subject to ophthalmic migraine. The remaining case reported by Junius (his case 3) seems to us to belong in an entirely different category. In this instance there were within the retina of each eye extensive white exudate around the optic disk and a massive white star figure around the fovea. The patient was aged 14 years. The only important similarity that the condition in this case had with that in the others lay in the fact that the exudate disappeared and there was little residual impairment of vision.

In 1935 Davis and Sheppard reported two cases as belonging to the type described by Junius. In one case the age of the patient was 23 years. "In each macular region was found an oval golden yellow area over which the retinal vessels passed." Visual acuity was 20/20—2 in each eye. Whether the lesion was elevated was not stated. Two years later, changes could still be seen in each macula, and vision of the right eye was 20/100 and that of the left 20/30. In their second case an elevated disciform lesion was observed in the right eye of a patient aged 41 years, who had angioid streaks in each eye. The observation was made about two years after central vision was said to have been lost. No improvement in vision was noted. It seems to us that the condition in this case belongs to the senile type of disciform degeneration, and we have therefore included it in the list of cases just analyzed. The assumption of Davis and Sheppard that the loss of central vision coincided with the onset of the disciform lesion is not necessarily correct, since sudden loss of central vision is a typical occurrence in cases of angioid streaks.

Junius regarded the lesions in his cases as retinal in origin and due to some vascular disturbance, but concluded that their etiology was still in the dark.

From the foregoing review of the literature it is obvious that the pathogenesis of senile disciform degeneration of the macula has not hitherto been determined. The literature relating to the juvenile type we have not yet fully examined, but thus far we have been unable to find in it any suggestive evidence as to the pathogenesis of the ocular lesions. The following cases studied by us seem definitely to reveal the pathogenesis of the senile, and strongly to indicate that of the juvenile, type of the disorder.

REPORT OF ADDITIONAL CASES

CASE 1.—*History.*—Miss G. L., aged 49, a white woman, first consulted Dr. Verhoeff on April 6, 1931, complaining that four days previously she discovered that the sight of the right eye was blurred.

Ocular Examination.—Vision of the right eye with a +2.50 D. sph. was 5/200, and that of the left eye with a +3.75 D. sph. ⊂ —1.00 D. cyl., ax. 125 was 20/15. On ophthalmoscopic examination of the right eye there was found occupying the macular region and extending farther below than above an almost black moundlike elevation with a smooth surface. Extending beneath the retina from the margin of the mound were several extravasates of blood. The fundus otherwise appeared normal—there was no sclerosis of the retinal vessels. The fundus of the left eye was normal.

General Condition.—The patient stated that her general health was excellent. Her urine was free from sugar and showed only an insignificant trace of albumin.

Course and Outcome.—A diagnosis of sarcoma of the choroid was made, and on April 9 the right eye was removed with the area under local anesthesia and a glass ball implanted.

On May 8, 1936, vision of the left eye with correction was 20/15, and the fundus was still normal. The general health of the patient remained excellent.

Pathologic Examination.—The eye was fixed in a 4 per cent solution of formaldehyde for forty-eight hours, and this was followed by immersion in acid alcohol for twenty-four hours and embedding in pyroxylin (celloidin). Serial sections showing the lesion in the fundus were made and stained with hematoxylin and eosin and with Verhoeff's stain for elastic tissue.

Microscopic Observations: The eye was normal except for the lesion in the macula and the presence of an unusual number of eosinophilic cells in the stroma of the iris. These cells (apparently eosinophilic myelocytes) were large, had small, round, solidly staining nuclei and were packed with fine eosinophilic granules. As many as five of these cells were sometimes observed in the high power field. The iris contained no plasma cells or other inflammatory cells aside from the eosinophils. The vessels of the retina and choroid and the posterior ciliary vessels were notably free from sclerosis. Peripheral cystoid degeneration of the retina was very slight. The supposed tumor of the choroid was seen to be a large mass of fresh blood beneath the retina in the macular region (fig. 1). This raised up the retina in the form of a mound measuring 1.5 mm. in height and 6 mm. in diameter. The mass was somewhat eccentric with reference to the fovea. It began 2 mm. to the temporal side of the fovea and extended to the margin of the disk. It reached its greatest height at a distance of 4 mm. from the disk, somewhat above the fovea. At least two-thirds the total mass of blood was beneath the pigment epithelium, which it had separated from Bruch's membrane in the form of a vesicle. The vesicle was solidly filled with blood and was 4 mm. in diameter. Its inner margin was 2 mm. from the margin of the optic disk, and its center was somewhat above the fovea. The vesicle was everywhere intact, except on the nasal side, where in a few sections it showed a rupture about 0.25 mm. wide through which blood had extended outside the vesicle between the retina and pigment epithelium. In the region of the fovea the wall of the vesicle was still in contact with the neuroepithelium of the retina in an area about 2 mm. in diameter, except immediately behind the fovea, where there was a slight amount of serum. In contrast to the blood within the vesicle, the blood outside of it contained lakes of serum free from blood cells, indicating that it was under less pressure than was originally the blood within the vesicle. The choroid, including the membrane of Bruch, was normal and intact except in a small area at, and just outside, the lower outer margin of the vesicle. Here the source of the hemorrhage was apparent (fig. 2). In an area 0.6 mm. wide the choriocapillaris with the membrane of Bruch was

EXPLANATION OF FIGURES 1, 2, 3 AND 4

Figure 1 (case 1) is the photomicrograph of a section showing hemorrhagic extravasate in the macular region beneath the pigment epithelium. The latter has been outlined with ink to show its position more clearly. In this section it was intact except for obviously artificial breaks. The blood outside of the vesicle extending to the margin of the disk left the vesicle through a small break in its wall seen in other sections. At the summit of the mound is a small amount of serum between the pigment epithelium and the retina proper. Magnified × 9. Figure 2 (case 1) is the photomicrograph of a section showing rupture of the choriocapillaris at the periphery of the mound, and the pigment epithelium elevated by hemorrhagic extravasate. Other sections showed this extravasate continuous with that forming the main mass on the left. Magnified × 48. Figure 3 (case 2) is the photomicrograph of a section showing the mound in the region of the fovea. The pigment epithelium in the posterior part of the mound is not the original pigment layer but has been newly formed. It is widely separated from Bruch's membrane by connective tissue. Magnified × 13. Figure 4 (case 2) is the photomicrograph of a section showing the nasal two thirds of the mound and large masses of old blood undergoing organization. The strands of pigment epithelium are taking part in the organization of the mass. Bruch's membrane is intact. Magnified × 44.

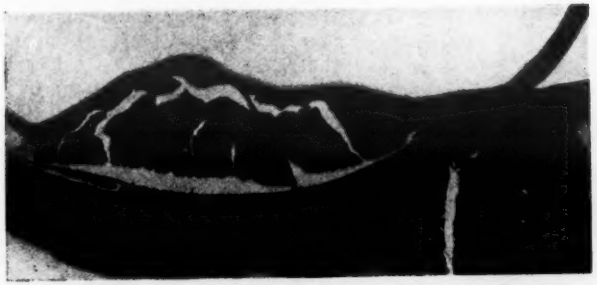


Figure 1

Figure 2

Figure 3

Figure 4

torn up in a ragged manner by an extravasation of blood which raised up the pigment epithelium in the form of small vesicles and then extended into the large vesicle described. From this point for a short distance beneath the large vesicle the choroid showed slight infiltration with lymphocytes and slight exudation of the latter cells into the mass of blood. The mass of blood here contained a slight amount of fibrin. From the margin of the vesicle outward the extravasate extended within the choroid just beneath the choriocapillaris for a distance of about 3 mm. Elsewhere the choroid was free from infiltration and hemorrhage. The retina proper was wrinkled in and around the fovea, probably as a result of the embedding process but otherwise appeared perfectly normal. It showed no exudation, hemorrhages or edema.

CASE 2.—*History.*—Daniel M., aged 77, a married white man, was admitted to the clinic of the Massachusetts Eye and Ear Infirmary on Jan. 15, 1936, complaining of blurred vision of his left eye.

He recalled a slight decrease of visual acuity of his left eye in the summer of 1934. He was certain that in 1935 vision of the same eye was much less clear than that of the right eye. In November 1935 he was told by an optometrist that his left eye was "stone blind."

Ocular Examination.—Vision of the right eye with a + 0.75 D. sph. was 20/30. Vision of the left eye was less than 20/200. Each eye was white and quiet. Arcus senilis was present. The corneas and other media were clear. The pupils reacted normally to light and in accommodation.

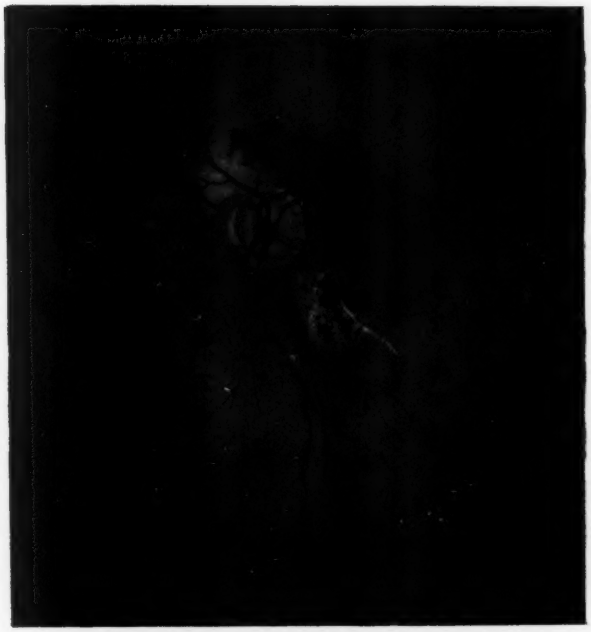
Examination of the Fundus.—The optic disk of the right eye was normal. In the macular region of this eye there were many colloid excrescences (Tay's choroiditis). The fovea was sharply outlined. The retinal vessels showed no definite sclerosis. The fundus was otherwise normal. The optic disk of the left eye was normal in color. The retinal vessels of this eye showed slight, if any, sclerosis. Adjacent to the temporal margin of the disk there was a large elevated mound, almost black, about 2 to 3 disk diameters in size, clearly demarcated and surrounded by a band of deep hemorrhage (color plate 1). The retinal vessels coursed in front of the mass and, with the exception of one vessel and its two branches, appeared normal. This vessel, which extended from the edge of the optic disk, became white as it crossed the mound, as did also its two branches. There were two diffuse whitish areas adjacent to the mass, one at its lower margin and the other at the upper border of the disk, each containing hemorrhagic spots.

At this time a definite diagnosis of hemorrhage behind the pigment epithelium was made.

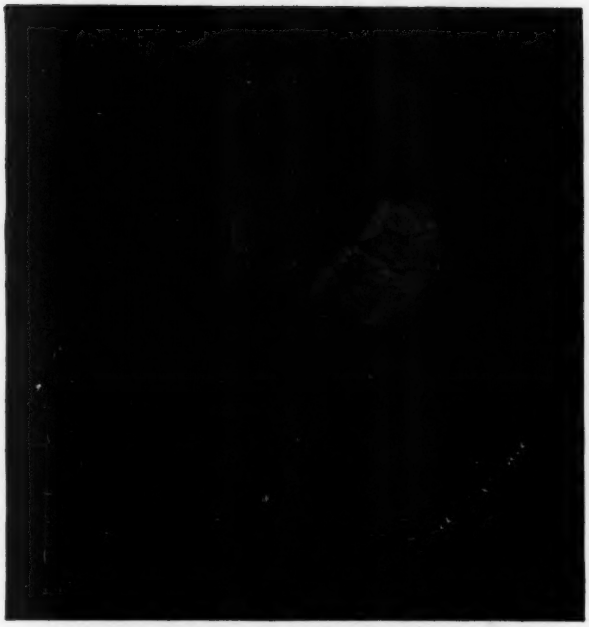
Physical Examination.—The blood pressure was 140 systolic and 80 diastolic. The heart was normal. The radial and dorsalis pedis arteries were not palpable. The urine contained no albumin, sugar or casts and rare red blood cells and white cells. The Wassermann and Hinton tests were negative. The blood count showed 75 per cent hemoglobin, 4,640,000 red cells and 8,000 white cells, with 63 per cent polymorphonuclears, 30 per cent lymphocytes, 1 per cent eosinophils and 6 per cent monocytes. The platelets were normal. Slight achromia and variation in size of the red cells were present.

Course and Outcome.—The patient was observed every two weeks during the next three months.

An area immediately adjacent to the temporal edge of the mound early became more shallow than the rest of the lesion and ultimately flattened out to the retinal level. The reflex from this portion became red. At the end of six weeks this



Color plate 1 (case 2).—Ophthalmoscopic appearance of the macular lesion when first seen. The picture is practically identical with that seen in case 1, except that in the latter case the retinal opacity below the mound and that above the optic disk were not present.



Color plate 2 (case 2).—Ophthalmoscopic appearance of the lesion three months later. The mound has become almost completely white. The hemorrhagic extravasates around it still persist, as do also the two retinal opacities. The latter were observed to be due to serum beneath the retina. The apparent minute retinal hemorrhages here were seen to be collections of red corpuscles in this serum.

portion of the mound seemed to disappear, leaving normal retina in its place except for a few deep hemorrhages.

Within four weeks the lesion became white in its temporal portion, gradually shading into gray and dark gray toward its nasal edge. The temporal edge was always sharply raised. At ten weeks the lesion was entirely white except for a small gray edge at its upper border. There were a few heavy granules of black pigment near its outer margin.

The lesion throughout its course remained completely surrounded by a band of deep subretinal hemorrhage. The white areas at the upper border of the disk and the lower border of the lesion persisted. They were raised at first, but finally became flatter, more pink and less distinct. In the vessels that originally appeared white as they crossed the mound, blood became visible in six weeks, and they then assumed the same normal appearance as the other retinal vessels. The final appearance of the lesion at the end of three months is depicted in color plate 2.

Three months after the patient was first observed, of his own volition he consulted two other ophthalmologists. Both these advised removal of the eye because it was practically blind and because of the possibility of a malignant growth. The patient was given our opinion that the condition was not malignant, but did not receive the positive assurance that he desired. He requested removal of the eye, and it was therefore removed, on April 15, 1936.

Pathologic Examination.—Preparations were made as in case 1.

Macroscopic Observations: The vitreous was clear and was not separated from the fundus or the lens. There were slight senile pigmentary changes at the ora serrata. In the macula there was an area 4.5 mm. in its greatest diameter, elevated about 1.25 mm. It was roughly circular but showed some indentations of the margin. Its surface showed slight grayish mottling and, in the center, the normal yellowish color of the macula, but otherwise appeared almost white. Around the area was a dark border from 0.25 to 0.75 mm. wide. Radiating from near the center were many fine white lines (artificial wrinkles?) which extended into the dark border. The retina otherwise was in situ everywhere.

Microscopic Observations: Aside from the lesion in the macula, the eye showed no pathologic changes and unusually slight senile changes. The ciliary processes showed only slight hyaline change. The two layers of epithelium of the pars plana showed fairly marked proliferative changes in some sections. There was almost no peripheral cystoid degeneration of the retina, and colloid excrescences were not seen anywhere. An unusual observation was that the myelinization of the fibers of the optic nerve ended about 1 mm. behind the lamina cribrosa. The optic disk was normal. The vessels of the iris, ciliary body and retina and the posterior ciliary vessels were normal. The vessels of the choroid were also notably free from endovasculitis, although an occasional artery showed slight intimal proliferation—less than would be expected, considering the age of the patient. The lesion in the macula (fig. 3) was seen to consist of a mound of finely fibrillated connective tissue permeated with blood and containing large masses of blood in the process of organization. It was situated between the retina proper and Bruch's membrane, was thickest behind the fovea and gradually became thinner towards the periphery all around. It reached to about 0.5 mm. from the margin of the optic disk. The new tissue was everywhere closely applied to Bruch's membrane, which, except for a few small defects to be described, was intact beneath it. The tissue in places was stained lightly by eosin but more often was stained slightly bluish by hematoxylin, especially near the choroid. The largest masses of blood were situated in the upper part of the mound and were nearer the retina than the choroid. In sections they comprised about half the

EXPLANATION OF FIGURES 5, 6, 7, 8 AND 9

Figure 5 (case 2) is the photomicrograph of a section showing a vein leaving the mound near its periphery and joining a large choroidal vein through a break in Bruch's membrane. Magnified × 54. Figure 6 (case 2) is the photomicrograph of a section showing an artery entering the mound from the choroid through a break in Bruch's membrane. Magnified × 87.

Figure 7 (case 3) is the photograph of a section showing the pigment epithelium elevated in the form of a vesicle by serous exudate in the foveal region. Serum has exuded from the vesicle and collected beneath the retina proper. Magnified × 10. Figure 8 (case 3) is the photomicrograph of a section showing the vesicle near its periphery. It contains two collections of red blood corpuscles. Here there is beneath the retina a considerable amount of serum which has been exuded through the epithelial walls of the vesicle. Magnified × 9. Figure 9 (case 3) is the photomicrograph of a section showing formation of connective tissue between the pigment epithelium and Bruch's membrane at the periphery of the vesicle. This tissue is entirely mesoblastic in origin and derived from fibroblasts that have penetrated Bruch's membrane. Magnified × 45.

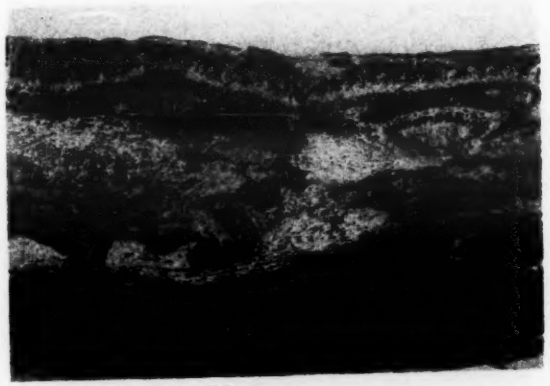


Figure 5

Figure 6

Figure 7

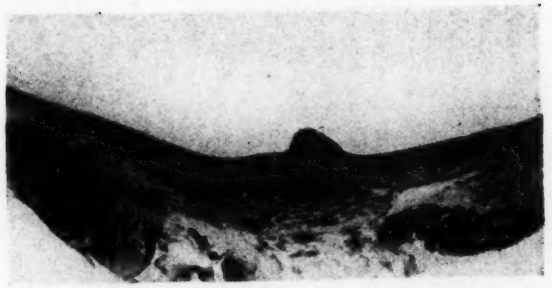


Figure 8

Figure 9

thickness of the mound (fig. 4). The tissue was fairly rich in fixed cells but was free from lymphocytes, plasma cells, pus cells and macrophages. It contained no hematogenous pigment, fatty acid crystals or cholesterol. The original pigment epithelium could be identified only at the periphery of the mound. In the central portion of the latter no pigment epithelium, new or old, remained adherent to Bruch's membrane or to the outer surface of the retina. At the periphery all around, connective tissue continued from the mound as a thin layer between the original pigment epithelium and Bruch's membrane for a distance of about 0.5 mm. As the single layer of pigment epithelium extended toward the center of the mound it was seen to have proliferated into two main layers which had diverged from each other and then further proliferated in an irregular fashion. Some sections showed a large central area of the mound entirely surrounded by a single layer of pigment epithelium. Posteriorly the latter had here undergone complete necrosis and appeared as an eosinophilic granular membrane free from nuclei but still containing pigment. In places, especially at the periphery, but also elsewhere, the pigment epithelium had proliferated in the form of irregular strands, some of which suggested acini. In the vicinity of these strands the connective tissue tended to assume a more hyaline character than elsewhere. Here there were also spindle cells, some of which contained more or less pigment, others being free from pigment. The epithelial origin of many of the latter was evidenced by their continuity or contiguity with the pigment cells and their close resemblance to the latter. From this evidence it was clear that a large proportion of the cells in the mound were derived from pigment epithelium. Many such cells were seen within the masses of blood and apparently were playing a large part in the process of organization. The mound contained a few thin-walled blood vessels, most of them slightly larger than capillaries, a few about three times this size. These were seen chiefly near the choroid and at the periphery. No vessels were observed entering the mound from the retina, but after careful search two fairly large vessels were noted extending into it from the choroid through sharp breaks in Bruch's membrane (figs. 5 and 6). One of these was directly connected with a large choroidal vein. In addition, a few microscopic breaks were noted in the membrane, through which fibroblasts extended into the mound from minute collections of newly formed fibroblasts in the choroid. In places Bruch's membrane was slightly wrinkled, evidently owing to contraction of the tissue forming the mound. Aside from these changes and slight compression, the choroid, including the choriocapillaris, appeared perfectly normal behind the mound, as it was elsewhere. The source of the hemorrhage was no longer recognizable.

The retina over the mound was relatively little altered. The nuclear layers, the ganglion cells and the layer of nerve fibers appeared normal. There were no hemorrhages, edema or exudation, and the vessels were normal. The outer surface was distorted and occasionally folded in the form of pseudorosettes. The rods and cones had been largely destroyed but were often still recognizable. The external limiting membrane was almost completely intact. As has already been stated, the pigment epithelium had completely disappeared from beneath the retina over the central portion of the mound, and the new connective tissue was directly applied to the neuroepithelium. In the fovea was a minute break into which the connective tissue extended.

Almost everywhere around the mound was a layer of blood extending between the retina and the pigment epithelium for about 1 mm. and on the nasal side reaching the margin of the optic disk. Into this blood many pigment cells had migrated. Unlike the blood within the mound, it was not undergoing organization.

Sections passing through the retinal opacity below the mound and others passing through that above the disk showed serum instead of blood between the retina and the pigment epithelium.

CASE 3.—*History.*—David W., aged 63, a married white man, was admitted to the clinic of the Massachusetts Eye and Ear Infirmary on Nov. 19, 1935, complaining of blurred vision in the left eye. He had noticed a black spot (probably of no significance) before the left eye for about five months and double vision with the eye for about two weeks. He had always been well except for measles in childhood and influenza in 1915. The family history was unimportant.

Ocular Examination.—The right eye was normal and had vision of 20/15 with a + 2.00 D. sph. ⌒ + 0.75 D. cyl., ax. 90. The optic disk showed an unusually deep and wide physiologic cup. The vision of the left eye with the glass the patient was wearing was 20/100, but with a + 1.00 D. sph. added it was 20/30. The retina showed in the macula a grayish elevation about the size of the disk. The retinal vessels were normal. The disk was cupped as in the right eye. There were no retinal hemorrhages. The vitreous was clear. On November 22 vision of the left eye with a + 1.50 D. sph. added to the glass was 20/30. On December 13 vision of the left eye with a + 1.50 D. sph. added was 20/40. On December 21 vision of this eye with a + 2.50 D. sph. ⌒ + 1.25 cyl., ax. 90 was 20/40.

Physical Examination.—General physical examination made at the Massachusetts General Hospital, Jan. 21, 1936, gave negative results except for numerous small telangiectases of the skin and slight hypochromic anemia. The blood pressure was 160 systolic and 80 diastolic. The red blood cells numbered 4,390,000. The Hinton test was negative. Roentgen examination of the teeth and chest gave negative results. The Graham test of the gallbladder was negative. The tonsils and urine were normal. At one time a soft mass was felt in the abdomen, but this disappeared in a few days.

Course and Outcome.—On April 3 vision of the left eye with correction was 20/70. The retinal lesion had changed in appearance. Beneath the elevated retina a dark mass could be indistinctly seen. There were no retinal hemorrhages. The vitreous was clear. A diagnosis of probable sarcoma of the choroid was made. On April 10 vision of the left eye was 20/200. The eye was enucleated.

Pathologic Examination.—Fixation, embedding and staining were carried out as in cases 1 and 2. Serial horizontal sections showing the lesion in the fundus were made.

Macroscopic Observations: On opening the eye there was seen an elevated irregular area in the macular region about twice the area of the disk. There were no hemorrhages around it. The surface was uneven, possibly due to the fixation. The yellow color of the macula was still well marked.

Microscopic Observations: On microscopic examination the eye was seen to be normal except for the lesion in the macula. Peripheral cystoid degeneration of the retina was slight below, extending backward from the ora serrata only about 1.5 mm., but it was excessive above, extending here for a distance of more than 6 mm. behind the equator. At the equator it was most marked, and almost split the retina into two thin layers. The optic disk showed an unusually deep physiologic cup. The retinal, choroidal and posterior ciliary vessels were notably free from endovasculitis. The elevation of the retina in the macular region was seen to be due to a subretinal exudate of serum. About half of this was confined beneath the pigment epithelium, which was lifted up from Bruch's

membrane in the form of a flattened intact vesicle (fig. 7). The vesicle was somewhat eccentric with reference to the fovea. Over its central portion the pigment epithelium was still adherent to the retinal neuroepithelium, but elsewhere it was separated from it by serum. The vesicle was 2.2 mm. in its greatest width and 0.35 mm. in its greatest height. Outside of the vesicle there was more serum below than above. Just below the vesicle the retina was sharply elevated 0.8 mm. in an area 3 mm. wide, but the serum extended under the retina for a total distance of 7 mm. Outside the vesicle there were no blood corpuscles or fibrin. The upper half of the vesicle was also free from these elements, but in the lower half there were two globular masses of blood corpuscles, which in a few sections took up about one-half the area of the vesicle (fig. 8). There was here also a slight amount of delicate fibrin in the serum. The pigment epithelium forming the wall of the vesicle was generally only one cell in thickness, but here and there the thickness had been increased to that of two cells. It showed no break anywhere. Many of the cells were greatly distended with serum and projected toward the retina, indicating that serum had been exuded from within the vesicle through the epithelium. At the nasal side of the vesicle there was a layer of new-formed connective tissue extending over Bruch's membrane for about 1 mm. (fig. 9). The serial sections showed that it extended vertically also about 1 mm. Beyond the margin of the vesicle it extended between the pigment layer and Bruch's membrane, gradually diminishing in thickness and disappearing at a distance of about 0.5 mm. from the margin of the vesicle. It reached its greatest thickness, about 0.4 mm., within the vesicle, where it was not in contact with the pigment epithelium. The stroma of the new tissue was hyaline and extremely delicate. It was fairly rich in fibroblasts, which were most abundant at and near its surface toward the cystic cavity. It contained a few vessels, some of them slightly larger than capillaries, but no more than two in any one section. Bruch's membrane was intact except for two small microscopic breaks. Actively proliferating fibroblasts could be seen extending from within the choroid through the breaks, and a capillary was noted passing through one of the breaks. In a few other places fibroblasts could be seen penetrating through Bruch's membrane into the new tissue without producing definite breaks in the membrane. The choroid beneath the new tissue was otherwise normal, except that it showed a few lymphocytes along the walls of some of the veins and possibly slight distention of the veins. The retina proper was slightly distorted over the vesicle but otherwise appeared perfectly normal.

COMMENT

The ophthalmoscopic picture seen in case 1 and that seen early in case 2 were essentially identical and were similar to those described in the literature as occurring early in cases of disciform degeneration of the macula. Except for the two areas of retinal opacity shown at the periphery of the mound, color plate 1 depicts the lesion in case 1 almost as accurately as it depicts that in case 2. In case 2, in which the eye was not removed until three months later, the ophthalmoscopic picture was observed gradually to change into that typical of the later stage of this disease (color plate 2).

On microscopic examination in case 1 there was observed a rupture of the choriocapillaris and, extending from this, a large hemorrhagic extravasate which had separated the pigment epithelium from Bruch's

membrane in the form of a large vesicle (fig. 1). That such a condition could occur has apparently never hitherto been demonstrated or even considered possible. It is interesting that the capillary rupture (fig. 2) was not beneath the center of the vesicle but at the periphery of the latter and hence at the periphery of the macula.

In case 2 the histologic picture was essentially the same as that seen by many other observers, with the important exception that in this case masses of blood undergoing organization were still present and thus furnished conclusive proof that the new tissue resulted from organization of blood. It was clearly evident that this blood was originally largely confined beneath the pigment epithelium, as in case 1, but its exact source could not be located, owing, no doubt, to the later stage at which the eye was removed. In this case, therefore, the possibility cannot be excluded that the blood was derived from the choriocapillaris by diapedesis. Since, as just pointed out, in cases 1 and 2 the ophthalmoscopic appearances were essentially the same as those described in the literature as occurring at corresponding stages and since in case 2 both the ophthalmoscopic and microscopic observations were essentially the same as those described in later stages of the disease, it is reasonable to conclude that hemorrhage beneath the pigment epithelium is the usual cause of senile disciform degeneration of the macula.

In case 3 there was seen a condition apparently not hitherto observed microscopically or ever recognized ophthalmoscopically, namely, a serous exudate occurring primarily beneath the pigment epithelium behind the macula and elevating the retina here in the form of a mound (figs. 7 and 8). From the standpoint of pathogenesis, the condition in this case was evidently closely related to that in the other two cases, but it is unlikely that the process would have finally resulted in the clinical picture of senile disciform degeneration. It is true that at the time the eye was removed there was definite proliferation of connective tissue on the surface of Bruch's membrane within the vesicle, but it is improbable that this would have gone on to the formation of a mass of connective tissue ophthalmoscopically conspicuous.

Clinically, also, the condition in case 3 differed from that in the other two cases in two important respects, namely, in the ophthalmoscopic picture and in the visual impairment. At the outset, in the macula there was a moundlike elevation darker than the surrounding retina but much lighter than that seen early in the other two cases. At this stage visual acuity was only slightly impaired in case 3, whereas in cases 1 and 2 central vision was almost, if not completely, abolished. The preservation of central vision for a time in case 3 was no doubt due to the fact that the pigment epithelium remained in contact with the rods and cones. Later, as was revealed by the microscopic examina-

tion, serum exuded through the epithelium and separated it from the rods and cones, thus causing loss of visual function here. At this stage there was seen with the ophthalmoscope what appeared to be a darker mass beneath the retina. This, no doubt, was the pigment epithelium separated from the retina proper, but it was mistaken for a sarcoma and led to removal of the eye. The early loss of central vision in cases 1 and 2 was probably due to the rapid and forcible elevation of the macula by hemorrhage and to the resulting distortion. It is noteworthy, however, that in each of these cases the pigment epithelium remained in contact with the fovea, although it was separated to some extent from the retina elsewhere beneath the macula by blood which had broken through at the periphery.

Owing to the age of the patient, 63 years, the condition in case 3 would not ordinarily be regarded as belonging to the juvenile type of disciform degeneration of the macula. It is not unreasonable to suppose, however, that in any patient free from arteriosclerosis a macular lesion could occur identical with that occurring in young persons. Certainly the microscopic characteristics of the lesion in this case seem to accord with the ophthalmoscopic appearances of the lesions in the juvenile type of disciform degeneration and also well explain the restoration of vision, with or without residual macular changes, that occurs in this disorder. For it is obvious that if in case 3 the serous exudate had become absorbed and the slight formation of connective tissue had ceased, visual acuity would have been largely, if not completely, restored. Moreover, the long preservation of central vision in case 3 and the absence of hemorrhage in the early stage are characteristic of the juvenile, not of the senile, type. Perhaps there will be found occurring at various ages, instances in which the character of the lesion is intermediate between that of the senile and that of the juvenile type.

Another condition that must be considered in connection with case 3 is so-called serous retinitis. This is often central; in fact, it is only the central type that one of us (F. H. V.) has ever observed. In such cases a large central area of the retina, including the macula but often extending beyond it, becomes diffusely cloudy and may show a wavy appearance resembling ripples of sand on the seashore. Vision is markedly impaired at the onset, but the condition always subsides, with recovery of good vision. The cause of this disorder has never been determined, but the observations in case 3 suggest that it may be serous exudation from the choroid. In serous retinitis, perhaps, the serum is under less pressure, is slowly exuded through the pigment epithelium without lifting it up and then permeates beneath or into the retina.

From a histologic standpoint, our three patients showed several features of considerable additional interest. In case 1 Bruch's mem-

brane showed only one break, a small one at the site of the rupture of the choriocapillaris, from which the hemorrhage arose. In case 2 there were noted only a few breaks in Bruch's membrane. These were very small, and through two of them new vessels passed from the choroid into the mound (figs. 5 and 6). The other breaks were filled with fibroblasts. Evidently, however, most of the connective tissue cells in the mound were derived from cells which had penetrated Bruch's membrane without producing microscopically visible breaks.

In case 3 only a very few small capillaries were observed extending through Bruch's membrane. In this case the proliferation of connective tissue cells through Bruch's membrane without causing breaks could be definitely recognized, for small spindle-shaped accumulations of such cells were seen here and there on the inner surface of the membrane, and occasionally a cell was observed extending through the membrane. In general, there were no accumulations of cells beneath the membrane within the choroid, although in a few instances slight accumulations were seen. The largest of these comprised about thirty cells in the section. No evidence could be observed that the retinal vessels played any part in the process of organization in either case 1 or case 3. In case 3 it was evident that serum was transudating from within the vesicle through the pigment epithelium. The latter showed no breaks, even microscopically, but many of its cells were distended with serum, which often could be seen to have broken from the cells into the space beneath the retina proper. A noteworthy feature in case 2 was the absence of hematogenous pigment in spite of the large amount of blood still remaining in the tissue. In case 2 it was clearly evident that the pigment epithelium played an important part in the process of organization. For from the epithelium large strands of cells were observed extending into masses of blood, and all stages in the formation from pigment cells of cells free from pigment and indistinguishable from fibroblasts were observed. In other places also the origin of the cells of the stroma from pigment epithelium was evident. In many places, however, it could not be determined whether the cells were of epithelial or mesoblastic origin, although no doubt those closely associated with the blood vessels were mesoblastic. In case 3 the new tissue was almost, if not entirely, mesoblastic in origin. What caused its formation in this case is not clear. There was no blood in its vicinity at the stage at which microscopic examination was made, but possibly blood had previously been present here. There was no condition seen in the choroid to explain its formation.

A question that arises in connection with the histogenesis of these lesions is whether similar lesions do not occur elsewhere than beneath the macula. One of us (F. H. V.) has examined sections of a number of eyes in which there was extensive formation of connective tissue between

the retina and Bruch's membrane elsewhere than in the macula, tissue similar to that seen in the advanced stage of disciform degeneration, but, unfortunately, has no evidence as to its histogenesis. Probably such conditions would not be recognized clinically as analogous to disciform degeneration of the macula.

Another question is whether the condition is related to Coats' disease. One of us (F. H. V.) has had an opportunity to examine sections of the lesions in the latter disease in both the early and the late stages and has found that the new tissue is formed in the external part of the retina but not primarily beneath the pigment epithelium. In the early stages there is a large amount of fibrin and hematogenous pigment present. In the later stages, cholesterol crystals surrounded by foreign giant cells may be numerous. It would seem, therefore, that Coats' disease is essentially different from disciform degeneration of the macula. The former disease seems to be primarily retinal, the latter choroidal, in origin.

In cases 1 and 2, hemorrhagic extravasates extended beneath the retina from the periphery of the mass. The microscopic examination in case 1 showed that such extravasates resulted from blood breaking through the pigment epithelium at one or more places at the periphery. In case 2 there were seen ophthalmoscopically small hemorrhagic extravasates apparently not connected with the main mass. These were noted to be entirely subretinal, so that actually they must have been originally derived from the main mass of blood. No doubt many of the separate retinal hemorrhages depicted in recorded cases also had this origin. In case 2 the gray retinal opacities below the mass and above the optic disk were observed to be due to serum beneath the retina. This serum contained a few discrete collections of red blood cells. Since the retina showed no edema or hemorrhage, it is evident that this serum had separated from the main mass of blood, just as serum usually does from any collection of stagnant blood. Probably some of the peculiar appearances depicted in various recorded cases can be explained in this way.

The causes of the hemorrhages in case 1 and 2 and of the serous exudate in case 3 remain to be determined Behr has advanced the view that disciform degeneration of the macula is due to some alteration in Bruch's membrane which permits an exudate to occur beneath the retina. But to us this seems highly improbable. It is true that with advancing age certain changes occur in the elastic portion of Bruch's membrane, as was first pointed out by Verhoeff and Sisson, but these changes are not more marked in the macular region than elsewhere, and even when very marked they are not necessarily associated with any alteration in the retina or impairment of vision. The inner portion of the membrane may also undergo senile change such as is commonly

seen associated with colloid excrescences, but this also does not lead to exudation from the choroid. Behr stated that in his case Bruch's membrane was split into two layers at the margin of the lesion, but it seems to us more likely that a new cuticular membrane had been formed by the proliferating pigment epithelium here. In advanced cases such as his it seems remarkable to us not that Bruch's membrane was altered beneath the mass but that it was so slightly altered.

The observations in our cases clearly indicate that disciform degeneration is due to some disturbance, possibly only temporary, in the choriocapillaris of the macular region, since they show the primary condition to be hemorrhage or serous exudation beneath the pigment epithelium. Obviously, the cause of the vascular disturbance in the choroid could be the same or different in different cases. The fact that the disorder most commonly occurs in patients of advanced age suggests that general angiosclerosis is a predisposing factor or that some condition associated with angiosclerosis is often an important factor in producing changes in the choriocapillaris. However, the observations in our cases, as well as those in the cases recorded in the literature, afford no support for the view that general angiosclerosis is always the cause of the disorder. For in the recorded cases sclerosis of the retinal vessels has been mentioned as occurring in only twenty-five of one hundred and twenty-nine eyes, and in our cases there was no evidence of angiosclerosis, either local or general, aside from insignificant sclerosis of some of the choroidal vessels in case 2. On the other hand, the observations in our cases do not exclude the possibility of a localized vascular lesion of infectious or other origin, since obstruction of a small vessel in the affected region could have escaped observation in spite of serial sections. In none of our cases did the choroid behind the lesion show recognizable edema, congestion or, aside from the site of the rupture in case 1, hemorrhage. Edema and congestion, however, could have disappeared before the eyes were removed if the vascular disturbance had been compensated or had subsided.

The fact that disciform degeneration of the macula has been observed in cases of angioid streaks is in accord with the conclusions at which we have arrived. For choroidal hemorrhages are frequent in cases of the latter condition and indicative of vascular disturbances which might well lead to hemorrhage or serous exudation from the choriocapillaris behind the macula.

There are a considerable number of cases in which disciform degeneration of the macula and circinate retinitis coexist. These comprise a high percentage of cases of the former condition, but a low percentage of cases of the latter. It seems evident that the two conditions must have at least one important cause in common or that one causes the other in these cases. The lesions in circinate retinitis appear to be

entirely retinal and consist chiefly of deposits of lipoid substance within large spaces in the retina about the macula and the phagocytosis of this substance by macrophages. There are also often small retinal hemorrhages. Ophthalmoscopically recognizable sclerosis of the retinal vessels may be present but is often absent. There is no known systemic metabolic disturbance to explain the condition. Since lack of oxygen is a cause of deposition of fat in the tissues, it seems possible that disturbance in the capillary circulation of the macular region may be the chief cause of circinate retinitis, as it probably is of the retinal hemorrhages often observed in these cases.

The macula is a specially differentiated vascular bed, as is also the choriocapillaris behind the macula. Hence it seems likely that when capillaries of the choriocapillaris are affected in some special way, those of the macular region of the retina are apt to be similarly affected. For the same reason also, it would be expected that each eye would often be similarly affected, a conclusion that accords with the fact that disciform degeneration and circinate retinitis are often bilateral. Since the structure of the retina is entirely different from that of the choroid, similar vascular changes in them might well produce different effects, in the one hemorrhages and deposits of fat, and in the other transudation of serum or hemorrhage by diapedesis or rupture. The fact that only rarely have disciform degeneration and circinate retinitis coexisted in opposite eyes can be explained by assuming that when alteration of the choroidal vessels is more marked in one eye, alteration of the retinal vessels is apt to be more marked in this eye than in the other.

On the other hand, the possibility cannot be dismissed that in at least some of the cases in which circinate retinitis and disciform degeneration coexist in the same eye, the former has predisposed to, or actually caused, the latter. For in advanced cases of circinate retinitis the pigment epithelium shows marked changes, and it thus seems possible that the same deleterious influence which has produced these changes may cause hemorrhage or serous exudation from the choriocapillaris.

The foregoing reasoning suggests the possibility that senile pigmentary changes in the macula may predispose to disciform degeneration, but this possibility seems remote, because such senile changes may be extremely marked without leading to hemorrhage from the choriocapillaris. It is, however, in accord with the facts that disciform degeneration in one eye is sometimes preceded by pigmentary macular changes in the same eye and is often associated with such changes in the other eye.

In arriving at a diagnosis of disciform degeneration of the macula the chief considerations are the size and situation of the elevated mass, the texture of its surface, the absence of inflammatory signs, the absence

of extensive separation of the retina, the coexistence of circinate retinitis and the presence of subretinal hemorrhagic extravasates extending out from the periphery of the mass. Such extravasates existed in our cases 1 and 2, have been described in many of the reports of the previously recorded cases and are therefore of great diagnostic importance when present. In Coats' disease the patient is almost always younger and the lesion larger than in any case of disciform degeneration. If, however, as might conceivably happen, the lesion in Coats' disease was small and confined to the macula, it could not be certainly distinguished ophthalmoscopically from disciform degeneration. It is more important to distinguish the latter from malignant melanoma and thus avoid needless removal of the eye. A small malignant melanoma situated in the macula and closely resembling disciform degeneration seldom occurs. In only one instance in the Massachusetts Eye and Ear Infirmary has such a tumor been mistaken for disciform degeneration. In this case the tumor showed several hemorrhagic extravasates due, as was afterward determined by microscopic examination, to necrosis of the tumor. Although situated near the periphery, they did not extend from beneath the mass. The mistake in diagnosis was soon rectified and did not delay removal of the eye more than two months. Much more frequent is the mistaking of senile disciform degeneration for malignant melanoma. This mistake was made by the senior writer in case 1. At that time it was unknown that a hemorrhage beneath the pigment epithelium could occur and produce the appearance of a perfectly black mound in the macula. The experience afforded by this case enabled one of us (F. H. V.) immediately to make a correct diagnosis five years later, when early in the course in case 2 an almost identical ophthalmoscopic picture was seen. In case 3 a diagnosis of disciform degeneration was made at first and was later abandoned when the appearance of the lesion changed. This mistake probably could have been rectified if enucleation had been delayed longer. We now believe that a malignant melanoma so seldom ophthalmoscopically presents the appearance of disciform degeneration that in a case of suspected tumor removal of the affected eye should be delayed until no suspicion remains that the lesion is that of disciform degeneration of the macula.

SUMMARY AND CONCLUSIONS

The literature relating to disciform degeneration of the macula is reviewed, and eighty-four cases of the senile type, one hundred and twenty-nine eyes being affected, are analyzed.

The clinical, ophthalmoscopic and microscopic observations in three cases of macular disease are described.

The observations in two of these cases prove conclusively that a hemorrhagic extravasate can occur between the pigment epithelium and Bruch's membrane, undergo organization and produce an ophthalmoscopic picture typical of senile disciform degeneration of the macula.

That this is the usual, if not the only, pathogenesis of this disease is strongly indicated by a comparison of the observations in these cases with those of the cases recorded in the literature.

In the third case the microscopic examination revealed the pigment epithelium lifted up in the form of a large vesicle by serous exudate. It is suggested that such a serous exudate may be the cause of the juvenile type of disciform degeneration of the macula and that so-called serous retinitis may have a similar origin.

The extravasation of blood or serum beneath the pigment epithelium in disciform degeneration of the macula is due to some disturbance in the choriocapillaris. Since the disorder is most commonly a senile disease, localized angiosclerosis is probably the usual cause of this vascular disturbance. In certain cases, especially those of the juvenile type, the vascular disturbance may have been dependent on some other cause or causes yet to be determined.

The frequent association of disciform degeneration of the macula with circinate retinitis may be due to the fact that each occurs in specially differentiated vascular beds. The possibility cannot be excluded, however, that in some cases, at least, circinate retinitis causes disciform degeneration by producing hemorrhage or serous exudation from the choriocapillaris.

In a doubtful case of malignant melanoma of the choroid, removal of the affected eye should be delayed until no suspicion remains that the lesion is that of disciform degeneration of the macula.

BIBLIOGRAPHY

Pagenstecher, H., and Genth, C. P.: Atlas der pathologischen Anatomie des Augapfels, Wiesbaden, C. W. Kreidel, 1875; cited by Junius and Kuhnt.

Michel, J.: Ueber Geschwülste des Uvealtractus, Arch. f. Ophth. **24**:131, 1878.

Walker, C. H.: A Case of New-Growth in the Macular Region, Tr. Ophth. Soc. U. Kingdom **17**:4, 1897.

Batten, R.: Peculiar Symmetrical Swellings in the Macular Region Apparently Due to Subretinal Hemorrhage, Tr. Ophth. Soc. U. Kingdom **24**:127, 1904.

Possek: Ueber senile Macularveränderung bei Arteriosclerose, Ztschr. f. Augenh. **13**:771, 1905.

Oeller, J.: Atlas seltener ophthalmoscopischer Befunde, Wiesbaden, J. F. Bergmann, 1905; cited by Julius and Kuhnt.

Lawford, J. B.: A Case of Subretinal New Growth of Doubtful Nature, Tr. Ophth. Soc. U. Kingdom **31**:257, 1911.

Axenfeld, T.: Retinitis externa exsudativa mit Knochenbildung im sehfähigen Auge, Arch. f. Ophth. **90**:452, 1915.

Hegner: Retinitis exsudativa bei Lymphogranulomatosis, Klin. Monatsbl. f. Augenh. **57**:27, 1916.

Beatson, H.: Notes on a Case of Tumor in the Retina (Massive Exudation), Tr. Ophth. Soc. U. Kingdom **36**:345, 1916.

Holm, E.: Retinitis exsudativa externa, Klin. Monatsbl. f. Augenh. **59**:319, 1917.

Elschnig, A.: Tumorähnliche Gewebswucherung in der Macula lutea, Klin. Monatsbl. f. Augenh. **62**:145, 1919.

Knapp, A.: Subretinal Exudate Simulating Sarcoma of the Choroid with Anatomic Examination, Arch. Ophth. **48**:559, 1919.

Neame, Humphreys: A Case of Retinitis Circinata, Proc. Roy. Soc. Med. **16**:11, 1923.

Coppez, H., and Danis, M.: Rétinite exudative maculaire senile, Arch. d'opht. **40**:129, 1923.

Feingold, M.: Senile Exudative Macular Retinitis with Remarks on Similar Tumor Formation in a Case of Angioid Streaks, Tr. Am. Ophth. Soc. **22**: 268, 1924.

Cords: Scheibenförmige Maculadegeneration, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **45**:246, 1925.

Junius, P., and Kuhnt, H.: Die scheibenförmige Entartung der Netzhautmitte, Berlin, S. Karger, 1926.

Verhoeff, F. H., and Sisson, R. J.: Basophilic Staining of Bruch's Membrane, Arch. Ophth. **55**:125, 1926.

Ormond, Arthur W.: Four Cases of Disc-Like Degeneration of the Macula Lutea, Guy's Hosp. Rep. **7**:16, 1927.

Paul, L.: Choroiditis exsudativa unter dem Bilde der scheibenförmigen Entartung der Netzhautmitte, Ztschr. f. Augenh. **63**:205, 1927.

Cushman, B.: Disciform Degeneration of the Macula, Am. J. Ophth. **11**:720, 1928.

Weizenblatt, S.: Fall von scheibenförmiger Netzhautdegeneration bei 40-jährigem Patienten, Ztschr. f. Augenh. **65**:299, 1928.

Holloway, T. B., and Verhoeff, F. H.: Disc-Like Degeneration of the Macula, Tr. Am. Ophth Soc. **26**:206, 1928.

Barriére, V. A.: Disciform Degeneration of Central Region of the Retina, Arch. de oftal. de Buenos Aires **4**:223, 1929.

Junius, Paul: Erscheinungsformen und Ablauf der juvenilen Retinitis exsudativa macularis, Ztschr. f. Augenh. **70**:129, 1929.

Hanssen, R.: Zur Anatomie der scheibenförmigen Degeneration der Netzhautmitte, Ztschr. f. Augenh. **72**:360, 1930.

Pallarés, Juan: Degeneratio disciformis maculae luteae, Klin. Monatsbl. f. Augenh. **86**:201, 1931.

Behr, Carl: Ein weiterer Beitrag zur Anatomie und zur Pathogenese der scheibenförmigen Degeneration am hinteren Augenpol, Ztschr. f. Augenh. **75**:216, 1931.

Duynstee, H. P. M.: Degeneratio maculae luteae disciformis, Klin. Monatsbl. f. Augenh. **88**:511, 1932.

Adler, F. H.: Disciform Degeneration of the Macula (Junius and Kuhnt), Arch. Ophth. **10**:379 (Sept.) 1933.

Kahler, A. R., and O'Brien, C. S.: Disciform Degeneration of the Macula, Arch. Ophth. **13**:937 (June) 1935.

Davis, W. T., and Sheppard, E.: Juvenile Macular Exudative Retinitis (Junius), Arch. Ophth. **13**:960 (June) 1935.

LOCALIZATION OF CHANGES IN THE EYEGROUND AND PEILINGATION OF THEIR PROJECTIONS ON THE SCLEROTIC

REPORT OF A NEW METHOD

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The precise localization of pathologic changes or a foreign body in the eyeground is one of the important and difficult problems of ophthalmology.

At the Thirteenth International Congress of Ophthalmology (held in Amsterdam in 1929), in discussing the operative treatment of retinal detachment, Lindner, Igersheimer, Brückner, Löwenstein and others pointed out that the precise localization of retinal rupture and the touching of this place with a thermocautery are very difficult and that the operation for detachment of the retina cannot become popular among ophthalmologists so long as new methods are lacking for the localization of the ruptured place and for the finding of its projection on the sclerotic when operating.

It is equally important to be able to localize precisely a foreign body adjacent to the eyeground, particularly in the posterior section of the eyeball.

Professor Natanson found search for a foreign body in the vitreous with a magnet to be harmful, as the posterior shriveling of the vitreous may involve detachment of the retina. He pointed out that retinal detachment after operation will occur much less frequently if localization of the foreign body is made with more precision. This pertains both to roentgenologic localization and to localization of the foreign body near the membrane of the eye by means of the ophthalmoscope. The precise localization of such an intra-ocular body may permit its extraction diasclerally with a magnet, almost without involving the vitreous and without unnecessary traumatization of the neighboring retinal parts; this may greatly diminish the danger of the occurrence of retinal detachment in the future. There exists a relatively extensive literature on the localization of intra-ocular cysticerci, initiated by Alfred von Graefe. If it is remembered, in addition, that a tumor or an inflammatory focus in the eyeground which is seen ophthalmoscopically is not measured in

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clinical practice (in my opinion, only because sufficiently elaborated methods do not exist for such measurement and localization which are accessible for every ophthalmologist), it will be evident that it is necessary to bring clearness and precision to the study of the localization of an intra-ocular change (such as a retinal rupture or an inflammatory or atrophic focus) or of a foreign body or a cysticercus near the membrane of the eye.

The study of such changes in the eyeground consists of two parts, which are equally important for the ophthalmologist. The first part treats of the precise localization of the morbid focus in the eyeball with reference to definite points of the eye, in either of the coordinates, which can be expressed in degrees or in linear measures. The object of the second part is the study of the best methods of topographic localization on the sclerotic of the point where it is necessary to make an incision or a cauterization when operating. The immediate finding of these points on the sclerotic is called peilingation, the term used by Tzeekoolenko. In a third part a new method of localization and peilingation will be discussed.

In the first part of the study of the localization my aim is to define the coordinates of the morbid focus in the eyeball. These coordinates must be given both for the surface of the eyeground and for the exterior surface of the eye.

I. LOCALIZATION OF THE MORBID FOCUS IN THE EYEBALL

Methods of Definition of a Scotoma Corresponding to the Morbid Focus in the Eyeground.—A disadvantage of these methods is the impossibility of applying perimetry and campimetry in cases in which the affected eye has poor visual acuity. Besides, the dimensions of a scotoma correspond, for the most part, to a much larger area of the retina than that of the morbid focus. Donders was the first to suggest that such scotomas be examined. He devised a ready reckoner for the defining of the distance from the limbus of the affected part of the eye. But this table is far from complete, is insufficiently precise and has become obsolete.

Donders examined a scotoma, i. e., he defined its location, by means of the general perimetric method. Goldfeder suggested that the location of a scotoma be defined by means of fire point perimetry in the dark. For this purpose he used an electric ophthalmoscope as an object, placed in front of it a diaphragm with a small opening and moved the ophthalmoscope along the arc of the perimeter.

Haase defined the location of a scotoma with a campimeter. But, owing to the errors in his calculations, this method cannot be regarded as an accurate one.

Methods of Measurement of the Distances in the Eyeground in Conformance with the Diameter of the Disk (Papilla Diameter).—To this group of methods belong those evolved by Schmidt-Rimpler, Kleefeld and Gonin. They are based on the discovery, by the judgment of the eye, of how many diameters of the optic disk could be set in a row along a certain distance. Therefore, their exactness depends largely on the ophthalmologist's judgment by the eye, and the individual error may sometimes be considerable. According to Schmidt-Rimpler, the diameter of the optic disk is admitted to be 1.5 mm.; the distance of the temporal margin of the optic disk from the posterior pole of the eye, 3.5 mm.; the radius of the eyeball, 12 mm.; its circumference, 74 mm., and the diameter of the cornea, 12 mm. Therefore, the distance from the margin of the cornea to the posterior pole must be 37 mm. less 6 mm., or 31 mm.; the distance between the margin of the cornea and the margin of the optic disk along the temporal side is 34.5 mm., and that along the nasal side, 26 mm.

Of course, Schmidt-Rimpler's calculations are approximate, but one cannot obtain greater accuracy. The diameter of the optic disk is conditionally admitted to be 1.5 mm., but the anatomic measurements of many authors show that the optic disk may vary in diameter from 1.2 to 1.8 mm.

For better orientation when measuring the eyeground Kleefeld suggested that a mesh be placed on the magnifying glass and the divisions of this mesh be compared with the size of the optic disk.

For the localization of retinal ruptures Gonin suggested his own method, based on the same principle. When during examination in a passable light Gonin found that the retinal rupture flashed as a red light on the background of the detachment, he marked at the limbus with india ink the meridian he had found. Then he measured the distance from the ora serrata in papilla diameters. If the ora serrata cannot be viewed, the distance in papilla diameters is measured from the exterior point on the periphery, which is visible with the ophthalmoscope. Then the number of papilla diameters is multiplied by 1.5 mm., and to the product is added 7 mm. (for the nasal side) or 8 mm. (for the temporal side), i. e., the distance of the ora serrata from the limbus. Certainly the exactness of Gonin's method is problematic.

Methods Based on the Principle of Perimetric Localization (Ophthalmoscopic Perimetry).—All the methods of this group are based on the principle suggested by Alfred von Graefe. The angle lying between the optic line and the line drawn from the retinal morbid focus through the nodal point of the eye to the observer's eye is measured perimetrically. The incline of this line toward the horizontal plane is also reckoned. For this purpose Graefe used a special ophthalmoscope.

The method which Graefe proposed in 1882 for the localization of a cysticercus was modified by Lindner and again applied for the localization of retinal ruptures (in 1929). The arc of the perimeter is attached to a large Gullstrand electrical ophthalmoscope, and in its ocular are inserted crossed threads. Along the arc of the perimeter is moved an electric lamp (for fixation of the visual line). The ophthalmoscopic image of the yellow spot coincides with the crossed threads in the ocular, and that of the retinal rupture is obtained when the eye is turned. The angle lying between two lines, the guiding line (from the cross to the retina) and the optic line (from the lamp to the yellow spot), is measured. The meridian is reckoned in conformance with the position of the arc of the perimeter.

It is obvious that if one modifies Graefe's method, placing the fixation point in the center of the perimeter and letting the observer move the ophthalmoscope along the arc of the perimeter, one may see also the extreme periphery of the eyeground. Besides, if the affected eye has reduced visual acuity, one may, by using the concomitant ocular movements, attain the fixation of the central mark of the perimeter.

All authors who applied the perimetric principle of localization followed this procedure. Dupuy-Dutemps and Pollack proposed this rule in 1910. But these authors made an error, because they considered the number of the perimetric degrees equal to the number of the arc degrees on the retina. Having defined the number of degrees of ophthalmoscopic divergence on the arc of the perimeter, they reckoned in the following manner: The divergence on the perimeter equals n degrees, and the number of the arc degrees on the retina also equals n degrees; then according to the formula $\frac{2\pi r}{360}$ n the length of the arc from the yellow spot to the morbid focus was calculated. The semicircumference of the eyeball was considered as equal to 36.5 mm. But this could be true only when the nodal point was located in the center of the eye turning. On this account the reckonings by this method are incorrect. In proportion as the focal distance from the posterior pole increases, the error of the localization becomes much greater, and the difference may attain 30 degrees even on the equator of the eyeball.

Professor Levitzky and Professor Goldman obviated this defect in 1927. They found a relation between the visual angle and the central angle, the latter of which it is necessary to know in reckoning the length of the arc, that is, the distance between the posterior pole of the eye and the focus in the retina. These authors used Gullstrand's schematic eye as a basis for the reckonings.

But the value of this work was considerably diminished by the fact that these authors used for the dimensions of the eyeball a series of arbitrary alterations which involved a perceptible error.

However, the table by Levitzky and Goldman appears to come much nearer to the truth than any other such table.

Other authors used the same type of ophthalmoscopic perimetry (Lenz, E. G. Lasarev, Weve and Reitsch). Weve's substituting method is original. When the perimetric examination of the affected eye is performed, a model is placed on this eye constructed according to the constants of Gullstrand's schematic eye. From an electric ophthalmoscope a ray of light is projected in a formerly defined direction, and on the surface of the wall of this model appears a bright spot which indicates the location of the focus. The distance from the limbus is measured in millimeters directly on the model of the eye.

A Method for Localization of the Focus According to the Distance of the Three Points in the Field.—This method was proposed by Booshmich and Roschat independently of each other. It may be called a tangential method. The method devised by Booshmich is very simple. All the measurements are performed with the aid of two simple rulers, each 75 cm. long. During the examination these rulers are placed at right angles. One ruler is attached horizontally to a support, which is placed at a distance of 30 cm. from the eye to be examined, so that the upper verge of the ruler divides the cornea in two. The other ruler, the vertical one, is moved along the first ruler to the right and to the left. The observer is situated behind both rulers and examines the patient's eye with an ophthalmoscope. At the same time the patient's eye is fixed on a candle light or on a marking attached at the zero point of the horizontal ruler. As soon as the morbid focus in the eyeground is found by means of the ophthalmoscope, the vertical ruler is placed so that the ray of light which is projected from the ophthalmoscope to the patient's eye may fall on the upper end of this ruler. Then the readings showing in centimeters the distance of the vertical ruler from the zero point and the elevation in centimeters of its upper end above the horizontal surface are checked. Thus the distances of the three points in the field are measured: (1) that from the fixation point (30 cm.), (2) that from the point of intersection of the two rulers and (3) that from the upper end of the horizontal ruler. With the aid of rather simple calculations, based on the constants of Werbitzky's schematic eye, Booshmich found the distances of the morbid focus from the limbus and the horizontal meridian (along the chord) for the reckoning of every 5 cm. on both rulers. Thus with the aid of the Booshmich ready reckoner and two simple rulers it is possible to localize rather precisely the morbid focus.

Pavia takes photographs of the eyeground from the optic disk up to the morbid focus and with the obtained photopanorama computes the location of the morbid focus. This is a complicated method.

As soon as the morbid focus on the retina and its projection on the sclerotic are localized, that is, as soon as the coordinates on the eyeball are found by either method, the next problem is to find this point according to the coordinates of the live eye, to define exactly the place of the rupture and to perform the operation. As has already been stated, Tzeekoolenko called this preparatory determination peilingation, that is, the finding of the point according to the already ascertained coordinates.

Peilingation, according to the methods suggested up to the present, consists of two stages: (1) the marking of a point on the meridian of the eye corresponding to the place of the focus and (2) the measuring of the focal distance from the recognized anatomic point—the margin of the cornea. The first of these procedures is generally performed according to the judgment of the eye (Gonin, Odintzov, Levitzky and others). The second is performed in various ways. Gonin measured the distance from the limbus to the focus by means of a divider and put the legs of the latter along the guiding thread, which is fastened at two different points of one meridian and divides the cornea into halves. Odintzov knots the guiding thread at the limbus and then measures the necessary number of millimeters from the knot and cuts the thread there. The end of this thread shows the place of rupture. Amsler suggested that a set of special metallic indicators of various sizes be used (for measuring the distance from the limbus to the focus). Loginov and Booshmich solved this question rather differently. In the first place, the distance from the limbus along the horizontal meridian (the external or the internal rectus muscle) is measured; then from the ascertained point the distance of the focus from the horizontal meridian is measured up or down (vertically). Loginov does this by means of a silk thread on the sclerotic. Booshmich measures both distances on the chord by means of a divider. But none of these methods guarantees the precise peilingation of the focus in the eyeball.

Lindner and Tzeekoolenko suggested special instruments by the aid of which one may measure on the sclerotic the necessary number of millimeters from the limbus. Lindner suggested the use of a device made of gutta-percha paper (a ring meridian) but soon abandoned it.

Guist's instrument has a fault. The screw which fixes the arrow is located above the corneal pole; therefore, it is impossible to turn the eye too far. The eye cannot be hidden under the lids, as the instrument prevents this. This defect is present in a greater degree in Tzeekoolenko's instrument. Its vertical handle, which is placed above the cornea, is 6 cm. long. Hence it would appear that peilingation of the focus during operation on the posterior segment of the eyeball, on and behind the equator, would be difficult and sometimes impossible to perform.

II. RECKONING OF THE POINTS OF THE PROJECTION OF INTRAOCULAR CHANGES ON THE SCLEROTIC

The precise methods of localization and peilingation must satisfy the following conditions:

1. There must be faultlessness as regards the laws of physiologic optics. 2. It is necessary to take into consideration the influence of the variations (*a*) of the size of the cornea and (*b*) of the length of the optic axis. 3. The method must be simple and must not require complicated instruments. 4. The localization and the peilingation must not require much time either before or during the operation. 5. It must be possible to apply the same method for the localization and peilingation of various changes of the eyeground, from retinal rupture to changes due to a nonmagnetic foreign body near the walls. 6. The possibility of a wider application of such a method in cases of various

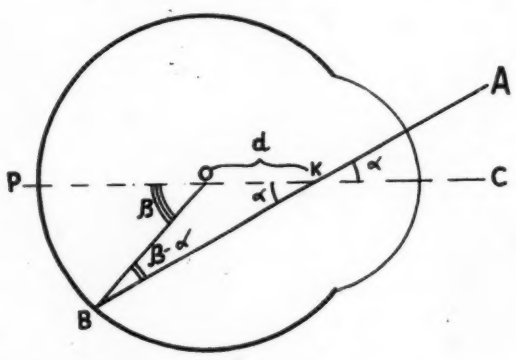

Fig. 1.—Werbitzky's reduced eye, illustrating the method of calculating the location of a morbid focus.

locations of the morbid focus in the eyeball (from the center to the extreme periphery) should be considered.

Beyond all question the method of ophthalmoscopic perimetry appears to be the most handy; hence most of the methods suggested up to the present day are based on this principle. In contrast to Lindner, who based his calculations on the eye, and to Listing, and Levitzky and Goldman, who used Gullstrand's nonprecisely schematic eye, I make all calculations according to the constants of Werbitzky's reduced eye.

If AB (fig. 1) denotes the direction of the ray of light from the examiner's eye, A to the morbid focus, B, then the angle on the perimeter between the line of fixation and the guiding line AB will equal the visual angle AKC.[1]

1. In all further calculations it is supposed that the angle AKC is corrected on the size of the γ angle, which must be precisely defined on the patient before starting the ophthalmoscopic perimetric study.

For the definition of the location of the focus, B, as regards the posterior pole of the eye, P, it is necessary to know the central angle β (POB). It is exterior to the triangle BOK and equals the sum of the two proximate angles, that is OKB and OBK. The angle OKB equals the visual angle α. The angle OBK (or the angle $\beta - \alpha$) can be easily reckoned from the oblique-angled rectilinear triangle OBK according to the well known axiom: The sides of a triangle coordinate as the sides of the opposite angles. If OK = d, i. e., the distance of the nodal point from the centrum, and OB = r,

$$\frac{d}{r} = \frac{\text{Sin } (\beta - \alpha)}{\text{Sin } \alpha}$$

$$\frac{d}{r} \cdot \text{Sin } \alpha = \text{Sin } (\beta - \alpha).$$

This is the basic formula for the calculations. According to the constants of Werbitzky's reduced eye, d is 6.4 mm., and the radius of the retinal curvature, r, is 10.2 mm. The equation then becomes:

$$\frac{6.4}{10.2} \cdot \text{Sin } \alpha = \text{Sin } (\beta - \alpha).$$

After the reckoning of this equation with the aid of mathematical tables the angle $(\beta - \alpha)$ is defined. By adding it to the angle α, the central angle β is obtained. During the further localization of the focus B, one may proceed in two ways. One may reckon on the sclerotic the distance (in millimeters) of the focus from the limbus, or one may define the distance of the focus from the anterior pole. The first mode is common to all methods without exception, and the second is the basis of the new method of localization and peilingation suggested now by me.

I shall discuss the first mode. One of the essential defects in reckoning the distance in millimeters is, to my mind, the lack of consideration of the variations in the corneal diameter. To define the influence of these variations I study the triangle ODL (fig. 2). The line DL represents one half of the diameter, and OL, the radius of the principal part of the eyeball from the centrum to the surface of the sclerotic. If it is admitted that the wall of the eye measures 1 mm. (the retina measures 0.4 mm.; the vascular membrane, 0.04 mm., and the sclerotic, 0.5 mm.), I assume that OL equals 11.2 mm. (10.2 + 1.0 mm). For the various possible corneal dimensions from 9.5 to 12.5 mm. (already mentioned), DL accordingly equals from 4.25 to 6.25 mm. From the rectangular triangle ODL is reckoned the correlation $\frac{DL}{OL} = \text{Sin } \delta$; for instance, $\frac{4.25}{11.2} = \text{Sin } \delta$, etc. With the mathematical tables one finds that the dimension may vary from 25.1 to 33.9 degrees.

These data are obtained if one admits that side by side with the wide variations of the corneal diameter the length of the radius of the eyeball (11.2 mm.) remains unalterable. But there is a strong pre-

sumption against this. Just as an average radius for the curvature of the surface of the eyeball was assumed, an average angle must also be chosen; that is, an average corneal diameter must be taken for the calculations. According to my measurements on six hundred eyes, the average length of the horizontal corneal diameter equals 11.13 mm. and that of the vertical diameter 10.36 mm. The arithmetical mean is 10.74 mm. As the average radius of the surface of the sclerotic (according to the constants of the reduced eye) I use the average size of the corneal diameter which I obtained with my measurements. Thus, in accordance with the equation $\frac{5.3}{11.2} = \text{Sin } \delta$, the angle δ equals 28.65 degrees. Next in order is the reckoning of the length of the arc B_1L, i. e., the distance of the projection of the focus on the sclerotic (B_1) from the corneal border. The arc B_1L is drawn together by the central angle ϵ, which equals 180 degrees $-$ ($\beta - \alpha$). I calculated the angle β after defining

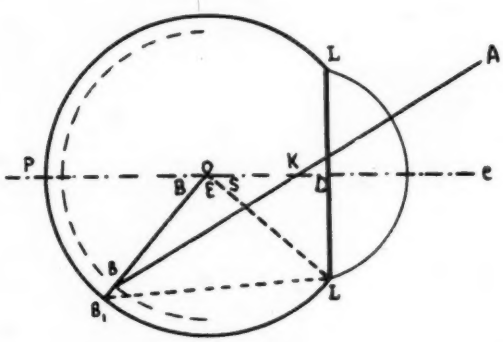

Fig. 2.—Reduced eye, illustrating the method of reckoning the angle δ.

the visual angle L; the size of the angle δ was obtained after measuring the corneal diameter.

When the size of the angle ϵ is known, one can reckon in millimeters the length of the arc B_1L in accordance with the formula $\frac{\pi K \epsilon}{180}$. Taking these calculations as a basis, I reckoned in millimeters a table of distances of the projection of the focus from the limbus in conformity with the size of the visual angle for every 5 degrees (table 1).

It must be borne in mind that all these calculations were made on the assumption that the length of the axis of the eyeball and the radius of its surface remain constant. But it is well known that they often vary. The lengthening of the diameter of the posterior half of the eyeball has a particular meaning in myopia. Lindner pointed this out and in cases of lengthening of the optic axis (in myopia) proposed to add 1 or 2 mm. to the reckoned distance from the limbus. However, this lowers the exactitude of the peilingation.

As for the variations in the radius of the surface of the eyeball, it is evident that every considerable alteration of its length involves considerable lengthening or shortening of the surface of the eyeball. In such cases the readings in millimeters on the sclerotic cease to be correct, and table 1, which shows precise reckonings only for the radius of 11.2 mm., loses its value. These data will be erroneous in cases of more or less considerable anatomic variations of the eyeball. In order to lessen, so far as possible, the influence of this factor on the exact solution of the problem under consideration, I elaborated a new method of localization and peilingation.

TABLE 1.—*Calculations in Millimeters of the Position of the Morbid Focus on the Sclerotic*

Visual Angle	Distance From the Posterior Pole of the Eye	Distance From the Limbus
5	1.6	28.0
10	3.2	26.1
15	4.8	24.8
20	6.3	23.3
25	7.9	21.7
30	9.4	20.2
35	11.0	18.6
40	12.5	17.1
45	13.9	15.7
50	15.4	14.2
55	16.8	12.8
60	18.2	11.4
65	19.5	10.1
70	20.7	8.9
75	22.0	7.6
80	23.1	6.5
85	24.2	5.4
90	25.2	4.4

III. NEW METHOD OF LOCALIZATION AND PEILINGATION

This method is very simple. The two coordinates of the projection of the focus on the sclerotic are denoted in degrees. Until recently all authors measured only one coordinate in degrees (the angle between the meridian on which the focus is located and the horizontal meridian), and the other coordinate was denoted in linear measures (millimeters), because it measured on the sclerotic the distance of the focus from the limbus. I denote the second coordinate in degrees as the distance of the focus from the anterior pole of the eye. Hence my method does not require many calculations. I determine by means of ophthalmoscopic perimetry what central angles (the distance in degrees of the focus from the posterior pole of the eyeball) correspond to the visual angles. The obtained angle, β, is deducted from 180 degrees, and one gets (180 degrees $-\beta$) the distance in degrees of the focus from the anterior pole of the eye (see table 2).

I named this instrument (see figure 3) the graduated peilingator (because its scale is divided in degrees), according to the new expression, peilingation, suggested by Tzeekoolenko. It consists of a thin metallic ring with a 14 mm. diameter of the clear space; the width of the ring is 3 mm. The instrument is placed on the eyeball concentrically to the cornea so that its interior surface clings close to the globular surface of the eyeball. On the exterior surface of this ring a small metallic rod is attached in such a way that its axis appears as a prolongation of the radius of the eyeball and forms an angle of 45 degrees with the optic axis.

In the upper part of this rod is a notch in which one may shift freely a small thin metallic arc; its direction may correspond to any meridian of the eyeball.

TABLE 2.—*Calculations in Degrees of the Position of the Morbid Focus on the Sclerotic*

Visual Angle, Degrees	Distance From the Posterior Pole of the Eye	Distance From the Anterior Pole of the Eye
5	8.1	171.9
10	16.3	163.7
15	24.4	155.6
20	32.4	147.6
25	40.4	139.6
30	48.3	131.7
35	56.1	123.9
40	63.8	116.2
45	71.3	108.6
50	78.7	101.3
55	85.9	94.1
60	92.9	87.1
65	99.7	80.3
70	106.0	74.0
75	112.3	67.7
80	118.2	61.8
85	123.7	56.3
90	128.9	51.5

Along the inner border of the ring, facing the limbus, at every 15 degrees are graduations; the axis of the inclined rod corresponds to the zero point of these graduations. A point is made with india ink at the upper end of the vertical diameter of the cornea, and one may then place the instrument on one of the coordinates of the morbid focus. The ring of the peilingator is placed on the eye concentrically to the cornea, and opposite the point, at 12 o'clock, is placed the line of graduation which corresponds to the meridian on which the morbid focus is located. With such placing the metallic arc which slides in the notch of the rod takes the direction corresponding to the meridian and will appear placed concentrically as regards the surface of the sclerotic. It will be everywhere at a distance of 2 mm. from the surface of the sclerotic.

The operator now pushes forward the anterior end of this arc in accordance with the graduations made on its marginal side, it being taken into consideration that the distance measured in degrees between

its end and the axis of the inclined rod will equal the value in degrees of the distance of the focus from the anterior pole (the second focal coordinate) reduced by 45 degrees (the distance in degrees of the axis of the inclined rod from the optic axis of the eyeball), and fixes it in this position by means of a screw.

The pointed indicator (the point of a pin) attached to the anterior end of the arc will almost come into contact with the sclerotic and

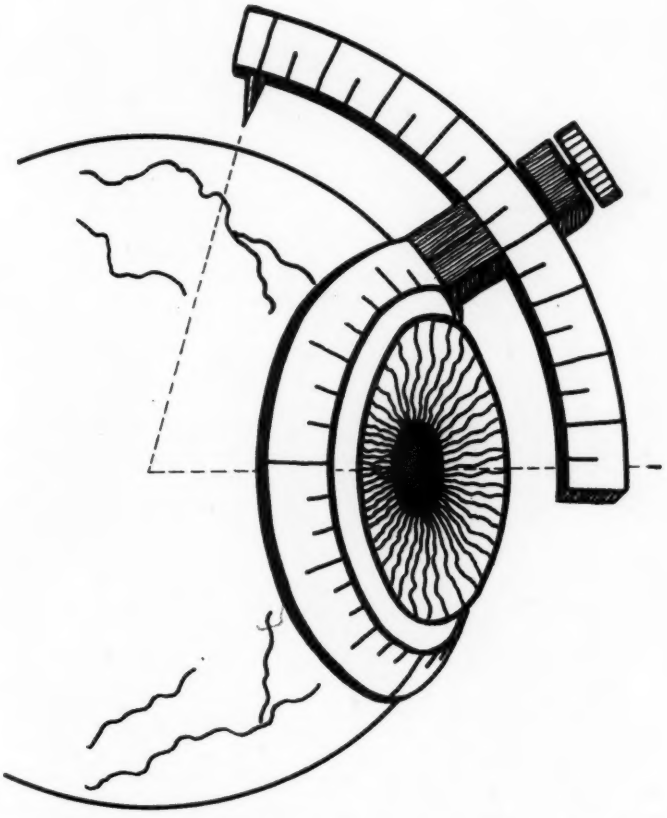

Fig. 3.—Drawing of Dashevsky's graduated peilingator. The scale is divided in degrees.

at the setting just mentioned will show the projection of the focus on the sclerotic.

The process of the peilingation of the point on the sclerotic consists of the following procedures:

1. A point is marked with india ink at the upper end of the vertical diameter of the cornea.

2. The sclerotic is laid open in the location of the focus.

3. The arc of the peilingator is fixed with a screw in a position which corresponds to the distance in degrees of the focus from the anterior pole of the eyeball.

4. The ring of the peilingator is placed on the eye concentrically to the cornea.

5. The assistant makes a mark with india ink on the sclerotic opposite the arc of the peilingator. That is the projection of the focus on the sclerotic and the exact place for the incision or the cauterization.

The apparatus is held in position on the eyeball by means of nippers which grasp the rod.

This rod will be in the region of the eyelid with any position of the eyeball. The ring of the instrument, which is not thicker than 0.85 mm., may disappear with the cornea behind the eyelids and will not hinder the rotation of the eyeball.

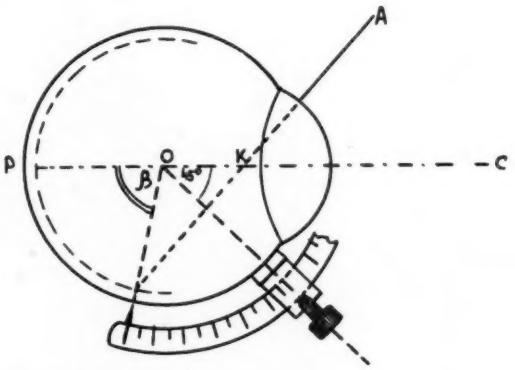

Fig. 4.—The graduated peilingator in place on reduced eye.

All the calculations for my new method are confined to the defining of the central angle, which corresponds to the visual angle, found by means of the perimeter. The first coordinate equals 180 degrees less this central angle. The second coordinate is the incline (in degrees) of the meridian on which the focus is located.

I shall now consider the helpfulness of the new method of localization and peilingation in obviating or reducing the aforementioned defect inherent in all the former methods and depending on alteration of the length of the eyeball. I shall analyze the same sample that Lindner analyzed. An eyeball with myopic refraction of 9.0 D. has an axis which is 3 mm. longer than normal. It is necessary to point out that in a person with myopia the change in the form of the eye is in the posterior segment only. Hence, if the focus is located at the equator of the eye its projection on the sclerotic will undergo no change which influences the precision of the peilingation. Two cases will now be con-

sidered. If it is supposed that the visual angle in the first case measures 30 degrees, then, according to the constants of the reduced eye, the central angle will measure 48.3 degrees. In that case the elongation of the radius will measure approximately 1.5 mm. (see figure 5). The distance from the center to the nodal point remains unchanged. The central angle B_1OP_2 is reckoned according to the sine formula

1. $\frac{OK}{OB_1} = \frac{\text{Sin } OB_1K}{\text{Sin } 30°}$
2. $\text{Sin } OB_1K = \frac{6.4}{11.7} \cdot \text{Sin } 30°.$

By aid of the mathematical table it is found that the angle OBK equals 15.9 degrees. Hence the central angle BOP equals 30 degrees + 15.9 degrees, or 45.9 degrees; i. e., because of the elongation of the axis of the eye it became diminished by 2.4 degrees (48.3 degrees — 45.9 degrees = 2.4 degrees).

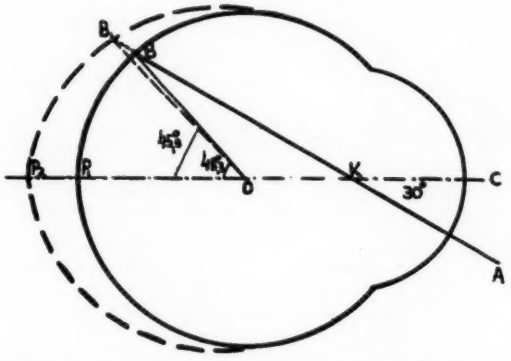

Fig. 5.—Diagram illustrating the method of calculating the central angle in a myopic eye.

Next, let it be supposed that in the second case the visual angle measures 15 degrees; i. e., the angle is located not far from the center. According to the constants of the reduced eye, the corresponding central angle will measure 24.4 degrees. In case of myopia of 9.0 D. the length of the radius will be approximately 3 mm. more and will measure 13 (10 + 3) mm.

The reckoning, made in the same way as in the former example, will show that the central angle measures 22.2 degrees; i. e., it will be diminished by 2.2 degrees. The nearer the visual angle is to the center, the smaller this difference will be, on account of the diminution of the central angle.

I am ready to consider the difference of 2.4 degrees as the highest possible one for my example. But the unit of 1 degree measures 0.25 mm. on the arc of the peilingator. Consequently, even on the arc of

the peilingator the error will not exceed 0.5 mm., but on the sclerotic this error will be much less, owing to the smaller length of the radius of its curvature. With the actual state of the surgical technic I take it for granted that the possible error will be 0.5 mm. I conclude that my method excels all others on account of these considerations and also because of the following: As was previously said, the six hundred corneas which I had measured varied in the vertical and horizontal meridians from 9.5 to 12.5 mm. Disregarding the variations of 9.5 mm. for the vertical diameter and 12.5 mm. for the horizontal diameter, which are rarely met with, I shall analyze the variations of from 10 to 12 mm. The coefficient of the correlation between the vertical and the horizontal diameter is not very great; it equals 31 per cent, but nevertheless it shows that when the one diameter is lengthened the other also has the tendency to become elongated. But what is going on in the other parts of the eyeball? One cannot answer this at present but can only make suggestions. Either the cornea becomes enlarged independently and the eyeball remains unchanged, or, in cases in which one sees a large or a small cornea, examination may show that the other parts of the visual organ have also undergone alterations as regards enlargement or diminution. The second hypothesis appears to be the more natural and probable one. It is indirectly corroborated by the results of the direct measurements. Tron made facometric observations on fifty-three emmetropic eyes and became convinced that the optic axis varies greatly. He found that 19.02 and 26.43 mm. are the extreme dimensions of the optic axis.

It is obvious that not only the length of the axis of the eyeball had been altered but that the eyeball as a whole was of greater or smaller dimensions in the various cases.

Hence it is also obvious that with such variations the length of the radius of the surface of the eyeball, on which all authors measure the distance from the limbus, also becomes altered. Because the slightest alteration of the radius results in the alteration of the length of the surface, one and the same distance in degrees will be shown by different numbers in millimeters, measured on the sclerotic.

But I must point out here that with the equal change of the size of the eyeball and of all its parts the correlation of these parts cannot be changed, and therefore the units in degrees remain unaltered.

The method that I have proposed is based merely on the measurement in degrees of the coordinates of the focus on the eyeground, and for the purpose of peilingation coordinates are taken which are shown in the angle units.

Let figure 6 be considered. The distance from the center of the graduated ring of the peilingator to the optic axis equals 7.92 mm. With

a radius of 11.2 mm. (i. e., 10.2 mm. long, according to Werbitzky + 1 mm. for the thickness of the wall of the eyeball) the angle between the optic axis and the axis of the rod of the peilingator measures exactly 45 degrees. Will it be changed if the length of the radius of the surface of the eyeball becomes changed? Let the extent of these changes be analyzed. In the first place, one must know the length of the radius that one can encounter. For a radius of 11.2 mm. I assume that the size of the corneal diameter is 10.74 mm.[2]

With the aid of simple calculations (based on the proportional changes of the corneal diameter and of the radius of the surface of the eyeball) I obtained the angles shown in table 3.

With the formula $\frac{7.92}{r} = \text{Sin I}$ reckoned the size of the angle δ between the optic axis and the axis of the rod of the peilingator. It

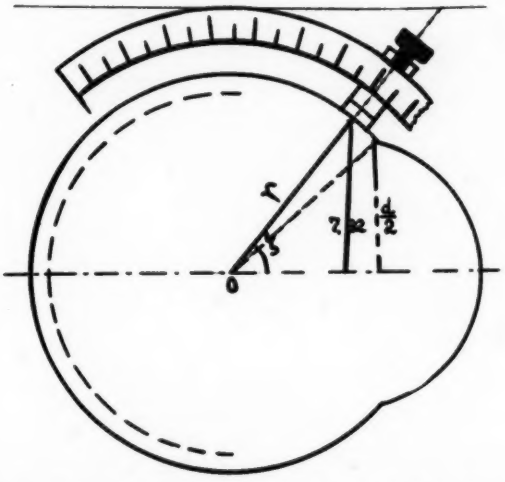

Fig. 6.—Diagram illustrating the method of calculating the inclination of the rod of the peilingator in accordance with changes in the radius of the eyeball.

appears that these changes are not great (see table 3). Considering that on the arc of the peilingator each degree equals 0.25 mm, and on the sclerotic it is much less, I am sure that my peilingator can be used with a correction when the corneal diameter varies from 10 to 12 mm. With a corneal diameter of 10 mm. it is necessary to regard the rod of the peilingator shifted to 50 degrees, with a corneal diameter of from 10.5 to 11 mm. to regard it shifted to 45 degrees and with a corneal diameter of from 11.5 to 12 mm. to regard it shifted to 40 degrees. This means

2. In accordance with my measurements the average length of the horizontal meridian equals 11.13 mm., and that of the vertical meridian 10.36 mm.; $\frac{11.13 + 10.36}{2} = 10.74$.

that with such variations of the corneal diameter the readings on the arc of the peilingator may be changed by 5 degrees. In other respects the process of peilingation remains unchanged.

The method of the localization and peilingation by degrees appears thus to be a unique method which permits one to take into consideration the different variations of the size of the visual organ.

The first case in which the reckonings in my table of the distances from the limbus and the new method with the use of the graduated peilingator were applied and verified corroborated their exactness.

REPORT OF CASE

G., a worker, had a trauma of the left eye six months before examination. The metallic foreign body remained in the eye during this period. The eye was quiet. Vision began to be reduced and was 0.2 on examination. Ophthalmoscopic examination showed a large metallic splinter, one end of which was firmly fixed in the wall of the eyeball at the ora serrata, the place of piercing. The other end

TABLE 3.—*Angles Obtained on the Basis of Proportional Changes of the Corneal Diameter and of the Radius of the Surface of the Eyball*

Corneal diameter (d), mm.	10.00	10.50	10.74	11.00	11.50	12.00
Radius of the surface of the eyeball (r), mm.*	10.43	10.95	11.20	11.47	11.98	12.51
Angle δ, degrees	49.40	46.30	45.00	43.70	41.40	39.30

* It is necessary to point out that when the angles obtained by me are compared as regards the extreme variations of the corneal diameter, namely, r = 9.03 for the diameter of 9.5 mm. and r = 13.04 for that of 12.5 mm. obtained by Tron, there appears to be a coincidence: The optic axis, according to Tron, varies from 19.02 to 26.43 mm., and according to my calculations 2r varies from 18.06 to 26.08 mm.

of the splinter hung loosely in the vitreous and was surrounded by flowing opacities. In the place where the splinter was attached one saw a white atrophic focus and a deposit of black pigment. Examination with the perimeter showed that the coordinates of the fixed end of the fragment were equal. 1. The visual angle (perimetrically determined) was 87 degrees. 2. The incline over the horizontal meridian of the eyeball was 10 degrees. The angle γ was positive and equaled 6 degrees. Subsequently the real visual angle (from the optic axic of the eyeball) equaled 87 degrees — 6 degrees, or 81 degrees. According to table 1, the distance from the limbus equaled 6.5 mm. By the method of Donders it equaled 9.5 mm., and by the method of Levitsky, 8 mm. It has been stated that the splinter was fixed at the ora serrata. The end of the ora serrata is generally 6 or 7 mm. from the limbus. Then were reckoned the coordinates for the graduated peilingator. The central angle (β) of 119 degrees corresponded to the visual angle of 80 degrees; hence the distance in degrees of the point sought for from the anterior pole of the eyeball was 61. When the arc of the peilingator was placed at 61 degrees it became evident that the distance of the point of the indicator from the limbus must equalize 6.5 — 7 mm. Thus, the measurements in degrees and the linear measurements, shown in my table, coincided.

On Nov. 25, 1935, an operation in which a magnet was used was performed (by Prof. D. M. Natanson). When the incision was made and the conjunctiva was separated the place where the wall of the eyeball was pierced appeared distinctly on the sclerotic as a dull and rather tender scar. The direct measurement

of its distance from the limbus was almost 7 mm. The point of the indicator of the graduated peilingator, placed on the eyeball, showed distinctly the place where the sclerotic was pierced. A small incision was made in this place, and by the aid of a hand magnet a metallic splinter 11 mm. thick and 7 mm. long was extracted.

Comment.—In this case ophthalmoscopic perimetry and natural control in the form of finding on the sclerotic a point sought for were successfully combined, and the advantage of the accurate reckoning based on the measurements of Werbitsky's reduced eye and of my precise method of localization and peilingation with the use of the graduated peilingator was distinctly shown.

BIBLIOGRAPHY

Booshmich, D. G.: Sovet. vestnik oftal. **5**:185, 1934.
Dashevsky: Tr. Ukrain. Leonard Hirshman Mem. State Ophth. Inst. **3**:151, 1935.
Fischer: Zentralbl. f. d. ges. Ophth. **25**:1, 1931.
Guist: Ztschr. f. Augenh. **72**:230, 1930; cited by Lindner, K.: Wien. klin. Wchnschr. **43**:225, 1930.
Goldfeder, A. E.: Arch. oftal. **1**:547, 1926.
Graefe, A.: Arch. f. Ophth. (pt. 1) **28**:187, 1882.
Haase, G.: Klin. Monatsbl. f. Augenh. **61**:324, 1918.
Lazarev, E. G.: Sovet. vestnik oftal. **5**:161, 1934.
Lenz, in von Elschnig, A.: Augenärztliche Operationslehre, Berlin, Julius Springer, 1922, vol. 2.
Levitzky: Russk. oftal. j., 1926, nos. 8-9, p. 825.
——and Goldman: Ukrain. med. Visti, 1927, no. 1, p. 17.
Lindner: Arch. f. Ophth. **123**:233, 1929.
Loginov: Russk. oftal. j. (nos. 2-3) **12**:192, 1930.
Lossev: Russk. oftal. j., 1928, vol. 9.
Natanson, D. M.: Sovet. vestnik oftal. **5**:41, 1934.
Odintzov and Orlov: Manual of Eye Surgery, Moskva Medgia, 1934.
Reitsch: Klin. Monatsbl. f. Augenh. **87**:145, 1931.
Report concerning the Thirteenth International Ophthalmological Congress, Russk. oftal. j. **11**:261, 1930.
Rochat, G. F.: Klin. Monatsbl. f. Augenh. **91**:411 (Sept.) 1933.
Salzmann, M.: Arch. f. Ophth. **123**:252, 1929.
Tron, Eugen: Arch. f. Ophth. **126**:454, 1931; Zentralbl. f. d. ges. Ophth. **25**:782, 1931.
Tzeekoolenko: J. nautshno-issledov. kafedri (no. 5) **2**:67, 1925; Russk. oftal. j. **11**:747, 1930; **13**:302, 1932.
Vasjutinsky, A. G.: Russk. oftal. j., 1926, nos. 8-9, p. 832.
Werbitzky: Russk. oftal. j. **8**:210, 1928.
Weve: Klin. Monatsbl. f. Augenh. **87**:145, 1931.

BILATERAL METASTATIC CARCINOMA OF THE CHOROID

REPORT OF A CASE

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NEW YORK

Metastatic carcinoma of the choroid and other ocular structures is of sufficiently rare occurrence to make the following report of interest. Carcinoma involving the intra-ocular and extra-ocular tissues is considered a secondary growth arising from a primary neoplasm situated elsewhere in the body. In about 70 per cent of such cases the primary growth is carcinoma of the mammary gland, other primary foci being the lungs, the stomach, the prostate and other organs. Sometimes the primary growth cannot be located during life and is revealed only by a general autopsy.

The ocular lesion may be the only visible evidence of metastasis; hence a systematic ocular examination should be performed in all cases of carcinoma. In this condition the media are clear, permitting a distinct view of the fundi. The characteristic finding is the choroidal tumor, which is flat and grayish. It is situated temporally around the posterior pole, where it is thickest, and sometimes extends from the ora serrata to and around the disk. It is covered and surrounded by a flat retinal detachment, which obscures the visibility of its contour. The carcinomatous tissue may invade one or several ocular structures, or these foci may occur independently of each other. Differentiation between them is usually impossible, even in serial sections of the eye.

Transillumination is frequently of no avail, owing to the posterior location of the growth. Tonometric examination is also of little value, since the tension is not increased until the occurrence of late complications. When the retrobulbar tissue becomes markedly involved there will be disturbed motility and exophthalmos.

The rapid development of the choroidal tumor causes detachment of the retina, resulting in a sudden marked deterioration of vision which in one fourth of such cases is bilateral. Simultaneous involvement of both eyes is rare; first one becomes affected and then the other. When the disturbance in vision is bilateral, a tumor can generally be recognized in each eye. It is usually impossible to differentiate between

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a unilateral flat carcinoma without a known primary growth and a flat sarcoma of the choroid. However, if clinically the growth suggests a flat sarcoma, enucleation in order to establish a diagnosis and prognosis should be considered.

The cancer cells are transported by the blood stream from the original focus to the ocular structures. These cells form emboli in the ocular vessels, and the emboli finally break through the walls of the vessels into the surrounding tissues.

Metastatic choroidal carcinoma occurs mainly in women, and the growth in both men and women usually appears between the ages of 30 and 60. The lapse of time between the occurrence of carcinoma of the breast and the appearance of the ocular manifestations is variable: it may be weeks or years. The outlook for life, even when the lesion is unilateral, is unfavorable; when it is bilateral the prognosis is even graver. In the former case death may be delayed for a few years; in the latter it is seldom delayed more than a few months.

Treatment is generally palliative, since the condition is a generalized one (carcinomatosis). In a very few cases radium or roentgen treatment has been tried with some success.

The typical pathologic change in the ocular structure consists principally in the presence of cancerous cellular elements. These are similar to those observed in the primary growth, but their arrangement varies according to the nature of the tissues which they invade or infiltrate. In the choroid the cellular type of carcinoma predominates: The cells replace the vessels of the choroid, arranging themselves in strands or cords, and small nests of epithelial cells form alveoli sparsely surrounded by fibrous tissue. The form of growth invading the retrobulbar region is usually the alveolar or scirrhous type of carcinoma. The sclera, optic nerve and sheath may be similarly involved, while the retina, iris and ciliary body are affected.

REPORT OF CASE

History.—F. J., a single woman aged 27 years, was admitted to the New York Skin and Cancer Unit of the Post-Graduate Medical School and Hospital on July 17, 1934, complaining of local pain caused by a mass in the left breast. The pain was first felt in September 1933. Her family history and her personal history had no bearing on the present condition. She was pale and obese. A complete physical examination revealed nothing abnormal with the exception of the condition of the breast. The right breast was normal. In the left breast a large mass could be felt in the outer half; it was hard, diffuse and somewhat infiltrated. Each axilla, but especially the right, showed enlarged nodes. A diagnosis of carcinoma of the left breast was made, and the patient was advised to enter the hospital for an operation. Radical mastectomy was performed on August 10.

The pathologic diagnosis of the removed tumor was scirrhous carcinoma of the breast (grade 2). Microscopic examination of sections of the breast showed a growth of epithelial cells arranged in nests and surrounded by a fibrous stroma (figs. 1 and 2). The nodes showed a similar structure (fig. 3).

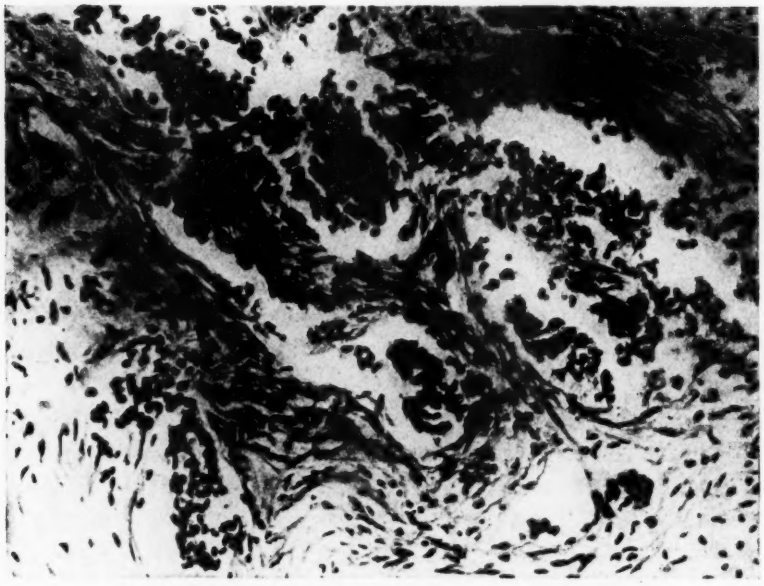

Fig. 1.—Photomicrograph (medium high power magnification) of the tumor of the breast, showing nests of tumor cells separated by edematous fibrous tissue.

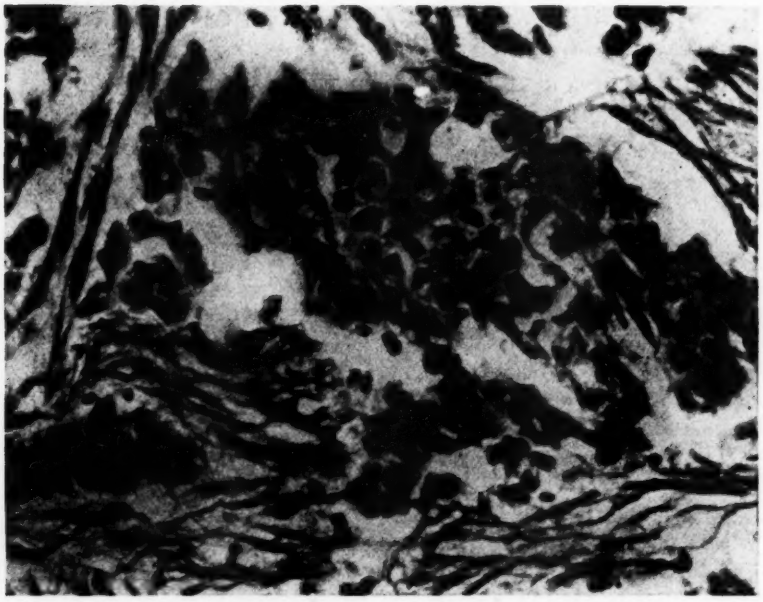

Fig. 2.—Photomicrograph (high power magnification) of an area from the tumor of the breast, showing large carcinomatous cells.

Ten days after the operation the wound became infected. The infection lasted four days, after which the wound healed. The patient remained in the hospital one month. During this time she complained of pain in the right sacro-iliac region, but roentgen examination of this area yielded negative results. After being discharged from the hospital she frequently returned for roentgen therapy. At her last visit to the clinic she complained of marked visual disturbances in each eye and was thereupon referred to the ophthalmic department of the New York Post-Graduate Medical School and Hospital.

Report of Ophthalmologic Examination.—The patient was admitted to the ophthalmic ward on Oct. 15, 1935. Four months before this date vision of each eye had suddenly become defective, first one eye being affected, then, a little later, the other. The visual disturbance was accompanied by general malaise, cough and pain in the right hip joint. Physical examination gave negative results, and the

Fig. 3.—Photomicrograph (medium high power magnification) showing metastatic focus of carcinoma in a regional lymph node, surrounded by lymphatic infiltration.

laboratory reported no abnormality. After a roentgen examination and study of the lungs, Dr. H. W. Meyer diagnosed the condition as a metastatic tumor secondary to a tumor of the lungs.

The external appearance of the eye was normal with the exception of moderate dilatation of the pupils, which reacted sluggishly to light and in accommodation. Vision was eccentric and was equal in the left eye to 5/200 and in the right to 3/200. The field of vision was faulty in each eye but could not be accurately determined, owing to lack of cooperation. No exophthalmos or loss of motility could be observed in either eye. In each eye the intra-ocular tension was normal, and the ocular media were clear.

The lesion in the fundus present in each eye was significant, consisting of a tumor in the choroid. The tumors in the two eyes were practically identical in

configuration, size, color and location (fig. 4). The mass was flat, sharply circumscribed, grayish and moderately pigmented. The tumor was covered by a flat retinal detachment, located posteriorly in the upper temporal and nasal area of the fundus. The choroidal growth in each eye was elevated 3 mm. The optic disk and retinal vessels appeared normal, and no hemorrhages or exudates were visible. The diagnosis of the fundic condition was bilateral carcinoma of the choroid (metastatic).

Course and Outcome.—During the patient's stay of one month at the hospital the aforementioned picture of the fundi remained practically stationary. She was discharged and transferred to the Bellevue Hospital, in the service of Dr. Webb Weeks, on November 27. I was given the opportunity of examining her frequently. After a short stay in the hospital she became cachectic, and the ocular condition became aggravated. In the right eye, vision was reduced to that in amaurosis. The pupil became widely dilated and fixed, the ocular conjunctiva chemotic and the fundus ischemic, and the retinal detachment seemed to be total. The tumor was now elevated 4 mm. The disk showed moderate papilledemas

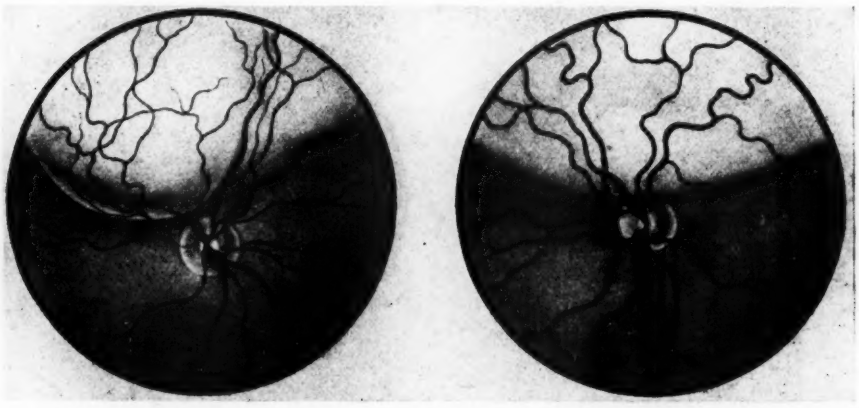

Fig. 4.—A drawing of the fundus of each eye on admission, showing flat bilateral retinal detachment due to bilateral metastatic carcinoma of the choroid.

surrounded by numerous small hemorrhages. The retinal veins became engorged and tortuous; the arteries became narrowed, and the intra-ocular tension rose to 56 mm. of mercury (Schiötz). The exophthalmometric reading was 20 mm., and the motility of the eye was restricted in all directions, causing pain on motion. The cornea, anterior chamber and media were clear. In the left eye, vision was reduced to perception of light, and there was faulty projection. The lids, cornea, anterior chamber and iris were normal. The pupil was widely dilated and fixed. Examination of the fundus revealed two large retinal detachments in the lower quadrant and a recent one in the upper temporal area. The circumscribed mass visible above the disk was elevated 5 mm. and retained its grayish color, but its outline was less distinct. No hemorrhages or exudates were present. The disk was hyperemic and elevated, indicative of incipient papilledema. The media remained clear and the retinal vessels normal. The intra-ocular tension was 29 mm. of mercury (Schiötz), and the exophthalmic reading was 18 mm. The diagnosis of the fundic condition was bilateral carcinoma of the choroid (metastatic), with bilateral retinal detachment and moderate bilateral papilledema.

During the patient's four month stay at the hospital several roentgen examinations were made, which showed metastatic foci in the lungs, skull, ribs and long bones. She gradually became extremely cachectic, and died on March 30, 1936. The diagnosis was carcinomatosis with metastases to the eyes.

Permission for a general autopsy was refused, but removal of the eyes was permitted. These were fixed and stained in the usual manner and were then sent to me through the agency of Dr. Edward Gresser.

Report of the Enucleated Eyes.—Right Eye: The eyeball was misshapen owing to manipulation following enucleation. Low power magnification (fig. 5) showed the anterior segment of the eye to be normal. The retina was detached. The posterior segment showed thickening of the choroid, principally on its temporal side and most prominent around the posterior pole. The choroidal growth appeared

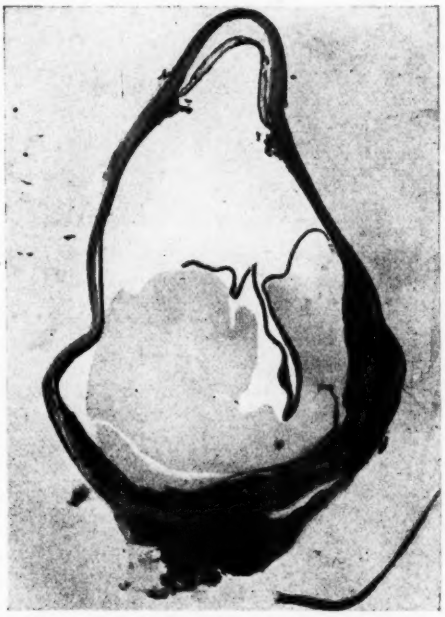

Fig. 5.—Photomicrograph (low power magnification) of the right eyeball, showing carcinomatous involvement of the choroid, sclera and retrobulbar tissue.

flattened and stretched from the disk to the region of the ora serrata. On the nasal side of the disk, extending anteriorly for a short distance, appeared a flat thickening of the choroid. The sclera showed a few dark spaces, probably cancerous foci. Surrounding the head of the optic nerve, at the apex of the retro-orbital region, were dark cancerous areas, which were more apparent on the temporal side in the neighborhood of the posterior short ciliary vessels. The head of the optic nerve, behind the lamina cribrosa, showed carcinomatous foci.

Microscopic study of sections of the eye revealed no lesions in the cornea, anterior chamber, iris or vitreous. The retina was completely detached, but its structural layers, including the lamina vitrea were normal. The important pathologic conditions appeared in the choroid (figs. 6 and 7). The vascular layers were replaced by carcinomatous tissues, arranged in acini which were filled with cancer cells. A few of their central areas showed degeneration, but no

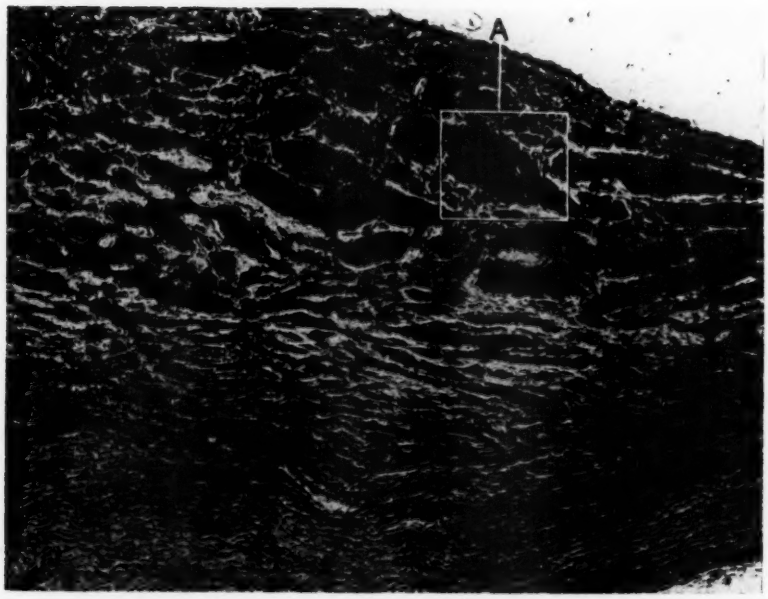

Fig. 6.—Photomicrograph (low power magnification) of the tumor in the choroid of the right eye, which resembles the tumor of the breast.

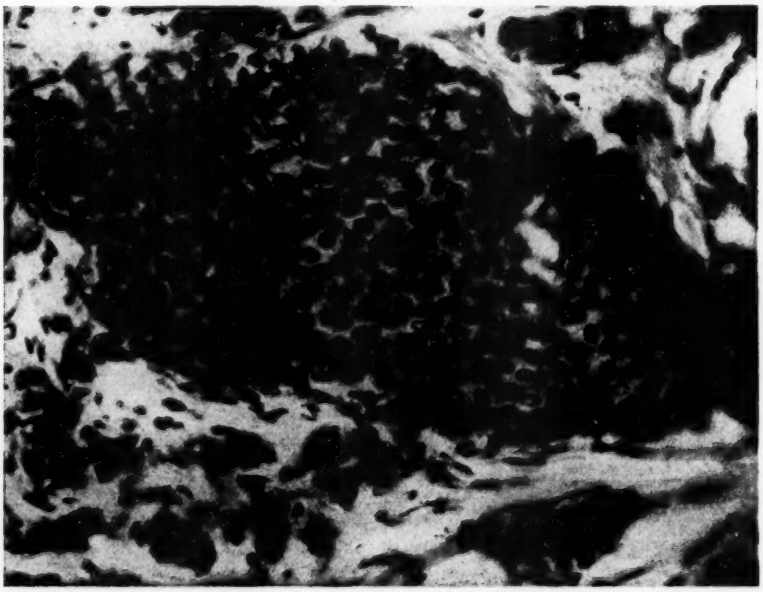

Fig. 7.—Photomicrograph (high power magnification) of area *A* of figure 6, showing slight degeneration in a cellular nest.

hemorrhages were observed. The arrangement of the cancerous tissue was mainly of the glandular type, owing to the structure of the choroid. There was some invasion of the lamina suprachoroidea. A few nests of cancerous cells were seen between the lamellae of the sclera. The stem of the fibers of the optic nerve just behind the lamina cribrosa showed a few nests of carcinoma cells, possibly transported by the central retinal artery. Surrounding the head of the optic nerve in the retro-orbital tissue (fig. 8) the cancerous invasion was marked, appearing in the vicinity of the posterior ciliary vessels. The arrangement of the cancerous tissue in this area was mainly of the alveolar type, owing to the nature and structure of the retrobulbar tissue invaded by the metastatic process. Degeneration of the cells was noted in the center of a few acini, but no hemorrhages were visible.

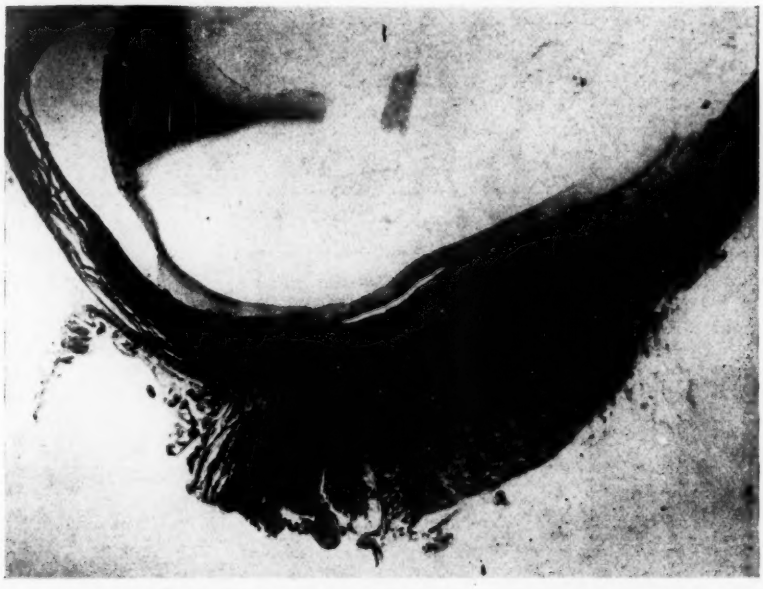

Fig. 8.—Photomicrograph (medium high power magnification) showing metastatic focus in the retrobulbar tissue and the optic nerve (right eye).

Left Eye: The eyeball (fig. 9) also was somewhat misshapen from manipulation. The media seemed clear. There was an artificial detachment of the retina. A thin mass could be seen encircling the posterior segment of the eye, and there was a thicker mass posteriorly in the retrobulbar region.

Microscopic examination showed nothing abnormal in the cornea, anterior chamber, iris and lens. The layers of the detached retina, including the pigment epithelium and the lamina vitrea, also appeared normal. The choroid was replaced by carcinomatous tissue, thinned out and extending from the ora serrata on the nasal side to the ora serrata on the temporal side. The flat growth was extremely cellular and apparently glandular in type. Some of the vessels in Sattler's layer were still recognizable. The choriocapillaris was completely replaced by carcinomatous tissue, and vessels contained carcinomatous cells (fig. 10). A few chromatophores were visible. There was some invasion of the lamina suprachoroidea, and the scleral spaces contained many nests of carcinomatous tissue. The retro-

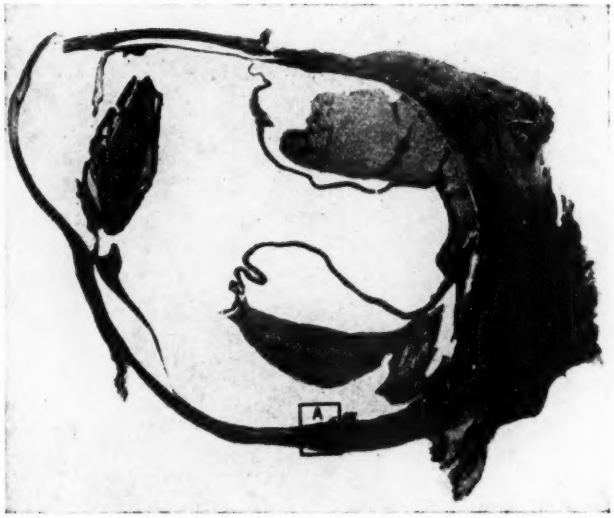

Fig. 9.—Photomicrograph (low power magnification) of the left eyeball, showing carcinomatous involvement of the choroid, sclera and retrobulbar tissue.

Fig. 10.—Photomicrograph (high power magnification) of area *A* of figure 9, showing an enlarged blood vessel containing carcinomatous cells.

orbital tissue surrounding the optic nerve head was markedly infiltrated with carcinomatous tissue of the alveolar type, and some of the smaller capillaries of this region were filled with carcinoma cells.

Pathologic Diagnosis.—This diagnosis was bilateral carcinoma of the choroid, with involvement of other ocular structures (metastatic).

COMMENT

Bilateral metastatic carcinoma of the choroid is rare; unilateral involvement occurs more often. In more than one fourth of the cases of the latter type the condition ended in bilateral ocular metastases.

Carcinomatous metastases may occur in one or several ocular structures, owing to an extension of the primary metastasis or to independent foci.

The visibility of the choroidal growth is often obscured by a flat retinal detachment. The finding of a primary carcinoma elsewhere in the body would suggest that the retinal detachment was caused by metastatic choroidal carcinoma.

An ophthalmologic examination is suggested in cases of primary carcinoma occurring in any part of the body, as the ocular lesion may be the only evidence of metastasis.

Further reports of ocular metastases are necessary in order to establish by statistics the relationship between these metastases and any primary carcinoma.

OCULAR DISTURBANCES ASSOCIATED WITH EXPERIMENTAL LESIONS OF THE MESENCEPHALIC CENTRAL GRAY MATTER

WITH SPECIAL REFERENCE TO VERTICAL OCULAR MOVEMENTS

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The region around the aqueduct of Sylvius is of special interest from an ophthalmologic point of view. One has to deal here not only with the nuclei of the third and fourth cranial nerves, below the anterior and the posterior quadrigeminal body, respectively, and with their afferent fibers for the optic and the vestibular reflex, but also with descending pathways, which transmit impulses from the hypothalamic centers of the vegetative system to the cervical portion of the sympathetic system and finally the tectal region and the nuclei of the central gray matter, the function of which is still rather obscure. Clinically it is known that lesions of the region induce anomalies of the pupillary innervation, disturbances of accommodation, and convergence, conjugate deviation of the eyeballs and paralysis of associated movements, particularly in the vertical direction. The pathologic processes producing these symptoms are, unfortunately, usually not sharply limited; they are tumors acting not only by localized destruction but also by pressure, the effect of which may reach as far as the base of the brain, or inflammatory processes extending to neighboring areas. Thus questions of finer localization may not be solved by study of clinicopathologic material alone; the pathologic changes observed in man raise questions rather than give an answer to these problems. It seemed, therefore, of interest to study the ocular symptoms in experimental stimulation and destruction of the region in the vicinity of the aqueduct of Sylvius.

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METHOD

The experiments were performed on thirty cats. As a rule, Clarke's stereotaxic apparatus was used for the stimulation experiments as well as for the production of circumscribed lesions. Since the brain was punctured from the dorsal aspect, in or close to the midline, in the region of the sagittal sinus, it proved advantageous to produce coagulation of this sinus by thermocautery after the skull was trephined above the sinus, so that the subsequent puncture was followed by a minimal hemorrhage only. In order to reach the central gray matter below the anterior quadrigeminal body the punctures were made in levels from 4 to 8 mm. cranial to the interaural line and reached from 19 to 23 mm. below their entrance into the surface of the hemisphere. An insulated silver wire with a bare tip was thrust into the brain, either in the midsagittal line or from 1 to 2 mm. lateral to the midline. The tip of the wire acted as the stimulating electrode, and a plate applied to the abdomen as the indifferent electrode. For the following electrolytic destruction the wire was connected with the anode of a source of direct current, and a current of from 1 to 3 milliamperes was applied for from two to three minutes. In a number of experiments lesions were produced by injection of small amounts (from 0.05 to 0.2 cc.) of a 70 per cent solution of alcohol or of a 20 to 40 per cent solution of formaldehyde instead of by electrolytic destruction. For this purpose a thin injection needle with a square end was placed in the stereotaxic apparatus instead of the silver wire. The injected fluid was stained with methylene blue (methylthionine chloride) or with eosin. After study of pupillary reactions and ocular movements for one or several days following production of the lesion, the frontal and the occipital center for ocular movements on each hemisphere were exposed and stimulated. In order to facilitate observation of the ocular reactions an incision was made into the external canthus, the nictitating membrane was excised, and the eyelids were widely separated. The extent of the lesions of the midbrain was studied on serial sections stained with Weil's stain or by the Loyez method.

COMMENT

Stimulation of the gray matter may yield constriction as well as dilatation of the pupil, the former symptom particularly if the electrode lies in the level of the posterior commissure and the latter symptom also if the more posterior parts are stimulated. Yet these observations should not be interpreted as indicating that the reflex arc of the pupillary light reflex takes its way over the mesencephalic gray matter, as some clinicians (Wilson [1] and Redlich [2]) supposed.

Spiegel and Nagasaka [3] showed some years ago (1926) that destruction of the gray matter around the aqueduct of Sylvius does not abolish the light reflex of the pupil. Ranson and Magoun's [4] stimulation experiments also indicate, as shown by their schematic diagram, that the reflex

1. Wilson, K.: J. Neurol. & Psychopath. **2**:1, 1921.
2. Redlich, E.: Wien. klin. Wchnschr. **35**:756, 1922.
3. Spiegel, E., and Nagasaka, G.: Arch. f. d. ges. Physiol. **215**:120, 1926.
4. Ranson, S. W., and Magoun, H. W.: Central Path of Pupilloconstrictor Reflex in Response to Light, Arch. Neurol. & Psychiat. **30**:1193 (Dec.) 1933.

arc of the pupillary light reflex takes its way outside the central gray matter, the pathway of the pupilloconstrictor fibers to the nucleus innervating the sphincter muscle arching around the central gray matter.

Thus the constriction of the pupil observed on stimulation of the outer parts of the central gray matter in the level of the posterior commissure is not a local symptom of stimulation of the gray matter but is due to stimulation of the surrounding pupillary reflex arc.

The pupillodilator effects observed on stimulation of the central gray matter are explained by the fact that the pupillodilator fibers originating in the hypothalamus descend in the ventral and the lateral aspect of the central gray matter around the aqueduct of Sylvius (Beattie, Brow and Long [5]). It should be emphasized that these stimulation effects are observed, as a rule, in each eye, even if the stimulating electrode has a paramedian position and a threshold stimulus is applied. This is in agreement with the view that the pupillodilator tracts (Karplus and Kreidl [5a]), as well as the constrictor pathway (Ranson and Magoun [4]), partially decussate peripheral to the point of stimulation.

After lesions of the mesencephalic central gray matter the pupillary light reflex may recover rather quickly, often on the first or second day after operation, even if the lesion is located in the region of transition between the mesencephalon and the diencephalon, the so-called pretectal area. Quantitative changes of the light reflex may, however, be observed, if the lesion reaches the vicinity of the sphincter nucelus or the afferent part of the optic reflex arc. Spiegel and Nagasaka [3] observed in cases of unilateral lesions of the anterior quadrigeminal bodies extending into the central gray matter a transient sluggish pupillary reaction on the side of the injury. Similar and more pronounced transitory changes of the pupillary reaction were observed in the present series of experiments. Not only was the reaction to light sluggish, but the contraction of the sphincter muscle continued for several minutes after the illumination of the eye had been discontinued. In other words, the reaction had a definitely tonic character. In this stage the pupils sometimes, under faint, diffuse illumination, were rather small (from 1 to 2 mm. in diameter). These findings may be interpreted in agreement with those of Magoun and Ranson,[6] who reported similar observations as due to a transitory stimulating effect of the lesion on the reflex arc in question, particularly since these phenomena

5. Beattie, J.; Brow, G. R., and Long, C. N. H.: Proc. Roy. Soc., London, s.B **106**:253, 1930.

5a. Karplus, I. P., and Kreidl, A.: Arch. f. d. ges. Physiol. **143**:109, 1911.

6. Magoun, H. W., and Ranson, S. W.: Central Path of Light Reflex: Study of Effect of Lesions, Arch. Ophth. **13**:791 (May) 1935.

were in our cases still more short lived (as a rule, from one to two days' duration) than in those of these authors, who observed a duration up to two weeks. We may add that psychic excitation produced by painful stimuli or electric stimulation of the frontal lobe could counteract this tonic contraction of the sphincter muscle but that its relaxation was somewhat less prompt and less pronounced than normally.

It seems unnecessary to emphasize that the permanent miosis observed in the Argyll Robertson pupil may not be explained in a similar way as these transitory phenomena. We refer in this regard to our previous discussion [7] of the Argyll Robertson pupil.

As to the innervation of conjugate ocular movements, it was shown in former experiments that after severance of the posterior longitudinal fasciculus (Spiegel and Tokay [8]) or after total transverse section of the brain stem behind the midbrain (Spiegel and Scala [9]) cortical stimulation no longer induces horizontal conjugate movements but induces only vertical conjugate movement or convergence movements of the eyeballs. These experiments showed that the essential mechanisms for the innervation of vertical ocular movements should not be sought in parts of the rhombencephalon, as supposed by some (for instance, the vestibular nuclei [Ohm [10]] or in the nucleus tecti of the cerebellum and the inferior olive [Muskens [11]]). While the cortical impulses innervating horizontal conjugate deviation have to take their way over the rhombencephalon, the connections of the forebrain with the mesencephalon proved to be sufficient for the cortical innervation of vertical ocular movements.

Continuing these experiments, we tried to determine the more exact position of the mechanisms that transmit the cortical impulses to the nuclear centers for the elevator and the depressor muscles of the eyeball. Clinically it is well known that paralysis of conjugate vertical ocular movements, so-called Parinaud's syndrome, occurs in diseases affecting the corpora quadrigemina or neighboring areas (the pineal gland, the

7. Scala, N. P., and Spiegel, E. A.: Pupillary Reactions in Combined Lesions of Posterior Commissure and of Pupillodilator Tracts, Arch. Ophth. **15**:195 (Feb.) 1936.

8. Spiegel, E. A., and Tokay, L.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **32**:138, 1930.

9. Spiegel, E., and Scala, N. P.: Cortical Innervation of Ocular Movements, Arch. Ophth. **16**:967 (Dec.) 1936.

10. Ohm, J.: Ztschr. f. Hals.-Nasen-u. Ohrenh. **39**:136, 1935.

11. Muskens, L. J. J.: (*a*) J. Comp. Neurol. **50**:289, 1930; (*b*) Deutsche Ztschr. f. Nervenh. **115**:81, 1930; (*c*) Das supra-vestibuläre System bei den Tieren und beim Menschen, Amsterdam, N. V. Noord-Hollandsche Uitgevers Maatschappij, 1934.

posterior part of the corpus callosum or of the thalamus and the cranial part of the pons). There exists, however, considerable difference of opinion regarding the interpretation of this symptom. Some have contended that symmetrical lesions in parts of the nuclei for the ocular muscles or of associational fibers may induce isolated paralysis of vertical ocular movements (Wilbrand and Saenger [12]) and that the assumption of the existence of supranuclear centers for these movements is not justified.

One may suppose, according to Spiller,[13] that certain fibers uniting parts of the oculomotor nuclei with one another or uniting the center for the inferior rectus muscle with that for the fourth nerve have a coordinating function similar to that of the posterior longitudinal fasciculus and that this function may be disturbed by small lesions. This view admits the existence of a coordinating system but leaves it undetermined where the fibers uniting the various parts of the nuclei for the ocular muscles take their origin. Holmes [14] considered it improbable that a nuclear lesion should destroy the cells innervating the elevator and depressor muscles on each side without affecting the other muscles. According to his view, this holds true particularly for the depressor muscles (the inferior rectus and the superior oblique muscles), which are innervated from two distinct nuclei separated by a relatively considerable space. Thus he arrived at the conclusion that supranuclear associational or coordinating mechanisms for up and downward movement must exist.

These supranuclear centers are generally supposed to be located in the neighborhood of the nuclei for the ocular muscles (Bielschowsky [15])—in the tectum, in the central gray matter around the aqueduct of Sylvius or in the tegmentum. Holmes inclined to the opinion that they lie in the anterior quadrigeminal bodies but arrived at no definite conclusion. Similarly, Riley [16] held that the superior colliculus controls vertical ocular movements, either through a center or through fiber pathways that pass through the colliculi. On the basis of studies in comparative anatomy, Muskens [11c] assumed that nuclei located in the gray matter around the aqueduct of Sylvius regulate the innervation of

12. Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 93.

13. Spiller, W. G.: J. Nerv. & Ment. Dis. **32**:417, 1905.

14. Holmes, G.: Brit. J. Ophth. **5**:241, 1921.

15. Bielschowsky, A.: Störungen im Augenbewegungsapparat, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 4, p. 173.

16. Riley, H. A.: Central Nervous System Control of Ocular Movements and Disturbances of This Mechanism, Arch. Ophth. **4**:640 (Nov.); 885 (Dec.) 1930.

vertical ocular movements. He distinguished a nucleus lateralis transmitting the impulses for downward movements and a nucleus medialis innervating the upward movements, but without proving that these nuclei have more importance for the mechanism of vertical movements than the centers postulated by him in the inferior olive and in the cerebellum. On the basis of the results of stimulation experiments Godlowsky [17] recently located the apparatus for upward movements and that for downward movements in two different regions, in the area of the posterior commissure and in the ventral part of the tegmentum, respectively. The nucleus of the posterior commissure he regarded as the supranuclear center for downward movements and the nucleus reticularis ventralis tegmenti as that for upward movements. Unfortunately, he studied only the effect of stimulation and the fiber degeneration of various tracts, without testing his theory by ascertaining the effect of subcortical lesions on the cortical innervation of ocular movements.

In our experiments stimulation of the central gray matter yielded downward movements of the eyeballs if the electrode was placed in the pretectal region or behind this level. The reaction appeared in each eye also if the electrodes lay lateral to the midline, indicating that the system that was stimulated had an effect on the homolateral and on the crossed nuclei for the ocular muscles. In about one half of the cases the downward movement was associated with dilatation, in the other half with constriction, of the pupils. Thus the supranuclear mechanism for these movements must be in the vicinity of the descending vegetative hypothalamic tracts as well as in that of the pupillary light reflex arc. From the point of view of finer localization, the combination of downward movement and convergence is of interest. It could be shown that the system stimulation of which with threshold currents induces downward movements lies dorsal to a point stimulation of which yields convergence movements. In cat 9 (fig. 1), for instance, stimulation at a distance of 21 mm. from the surface elicited downward movement of the eyes and dilatation of the pupils (the distance of the coils of the Harvard inductorium with two dry cells was 10 cm.); at a distance of 22 mm. we obtained horizontal inward movement of the right eye and inward rotation of the left eye, and at a distance of 23 mm. from the surface inward movement of both eyes. Several times the downward movement of the eyes was associated with movement of the head in the same direction, and in one case (cat 12) also with closing of the mouth. The first-mentioned combination seems of

17. Godlowski, W.: Les centers sous-corticaux du regard: Etude expérimentale, Krakow, 1936.

interest in view of the not infrequent clinical observations in epidemic encephalitis of a combination of oculogyric crises accompanied with tonic movements of the head in the same direction. One may suppose that the systems descending from the central gray matter or its neighborhood in the level of the posterior commissure not only unite the parts of the nuclei for the ocular muscles innervating vertical movements with one another but also reach the segmental nuclei innervating movements of the head in the same plane.

More rarely than downward movements, upward deviation was observed. This reaction appeared often combined with conjugate devia-

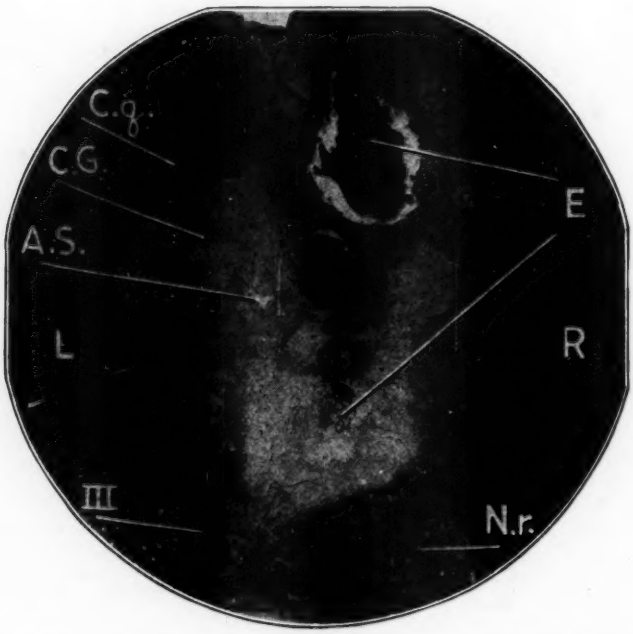

Fig. 1 (cat 9).—*E* indicates the puncture canal through the right corpus quadrigeminum (*C. q.*) and the right half of the central gray matter (*C. G.*), surrounded by the electrolytic lesions; *A. S.*, the aquaeductus Sylvii; *III*, the oculomotor nerve; *N. r.*, the nucleus ruber; *L*, the left side,, and *R*, the right side.

tion to the opposite side when the electrode was not moved far enough downward to reach the level of the aqueduct of Sylvius and medial parts of the tectum were stimulated and, particularly, when the electrode lay more anteriorly, reaching caudal parts of the optic thalamus. This is in agreement with the statment of Godlowski [17] that stimulation of medial parts of the anterior quadrigeminal body and of internal parts of the optic thalamus yields upward deviation of the eyeball. In this connection clinical observations of Holmes [14] are of interest; he reported that tumors or lesions extending from before backward into the midbrain affected first upward, then downward, movements and eventually

convergence, indicating that the center for elevation of the eyeball lies anteriorly and that for depression posteriorly.

The appearance of vertical conjugate movements on stimulation of the cortical frontal and occipital centers was prevented by producing lesions of the central gray matter in the most cranial part of the midbrain and in the region of transition to the optic thalamus. This may be illustrated by the results of the following experiments.

EXPERIMENTS WITH LESIONS OF CENTRAL GRAY MATTER IN REGION OF AQUEDUCT OF SYLVIUS

CAT 8.—On Nov. 30, 1936, at 12 o'clock, puncture with the stereotaxic apparatus was made 4 mm. anterior to the interaural line, 1 mm. to the right of the midsagittal line and 20 mm. below the surface. On stimulation (the distance of the

PROTOCOL 1.—*Cat 8*

Time	Stimulated Area	Ocular Reactions	Other Reactions
3:25	Left frontal lobe..... (distance of coils, 7 cm.)	Each eye deviated to right	Cloni of right hindleg
3:30	Left frontal lobe.....	Each eye deviated to right	
3:50	Left frontal lobe.....	Each eye deviated to right	Cloni of right half of body
3:54	Right frontal lobe.....	Horizontal undulation; dilatation of pupils	
3:58	Left frontal lobe.....	Left eye showed clonic jerks to right after stimulation ceased; right eye showed no definite movement	Cloni on right side
4:05	Right frontal lobe..... (distance of coils, 6 cm.)	Left eye showed clonic movements to left; right eye showed no definite movement	
4:08	Left occipital lobe.....	Each eye deviated to right	
4:10	Right frontal lobe.....	Each eye deviated to left	General cloni
4:13	Left frontal lobe.....	Each eye deviated to right	General cloni
4:15	Left occipital lobe.....	Each eye deviated to right	
4:18	Right occipital lobe.....	Each eye deviated to left	

coils being 10 cm.) there were movement of the eyeballs downward and contraction of the pupils. Electrolysis (with a current of 1 milliampere applied for three minutes) was carried out in this position, in the midline and 1 mm. to the left of the midsagittal line (the other indexes were the same). After electrolysis on the left side the electrode was inadvertently lowered from 2 to 3 mm.

At 3 p. m. there was a light reflex on each side. The right pupil contracted from 7.5 to 6 mm., and the left pupil from 7 to 6 mm.

At 3:25 p. m. stimulation of the cerebral cortex was carried out (protocol 1).

Histologic Examination.—The electrodes had penetrated the posterior commissure and produced lesions in the central gray matter below the commissure on each side (fig. 2). The oculomotor nuclei and the Edinger-Westphal nuclei were intact. On more caudal sections the left electrolytic lesion was continued by a fine canal that passed through the most lateral part of the central gray matter and reached the roots of the left oculomotor nerve. The majority of the fibers of this root were, however, intact. The right oculomotor nerve was normal.

CAT 14.—On Jan. 16, 1937, puncture was made 7 mm. anterior to the interaural line in the midsagittal line, extending to a depth of 21 mm. Stimulation (the distance of the coils being 8, 8.5 and 9 cm.) elicited upward movement of the eyes and dilatation of the pupils. Electrolysis (with a current of 1 milliampere applied for three minutes) was carried out in the midline and 2 mm. outside the midline on each side.

On January 19 the right pupil was wider than the left, and there were a distinct reaction of the left pupil to light and an uncertain reaction of the right pupil to light.

Stimulation of the left frontal lobe (the distance of the coils being 6 cm.) elicited repeatedly conjugate deviation of the eyes to the right. In some experiments

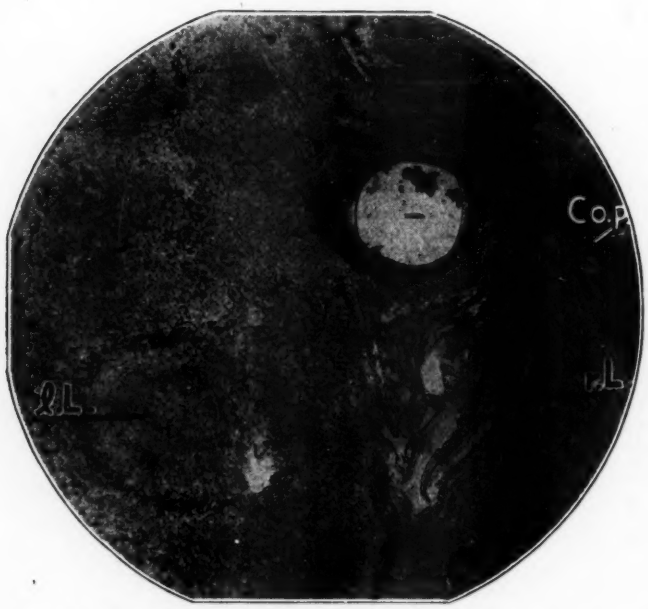

Fig. 2 (cat 8).—Electrolytic lesion in the left (*l. L.*) and the right (*r. L.*) half of the central gray matter below the posterior commissure (*Co. p*).

the head also turned to the right; in others there was downward movement of the head but no vertical ocular movement. Stimulation of the left occipital lobe elicited slight convergence movements.

Histologic Examination.—The maximal extent of the lesion was observed in sections through the caudal end of the third ventricle (fig. 3). Here the periventricular gray matter was destroyed between the ganglia habenulae and the corpora mammillaria. Forel's field H and the cerebral peduncles were intact; on the left side the lesion extended somewhat into the medial part of the optic thalamus and the left ganglion habenulae. The lesion was seen to diminish rapidly in more caudal sections, and was limited, on section through the posterior commissure, to the central gray matter around the aqueduct of Sylvius. The oculomotor nuclei and nerves were not involved.

In our cases in which vertical conjugate deviation failed to appear on cortical stimulation after lesions of the central gray matter, the oculomotor nuclei were intact or, at most, injured on one side. This could be proved by the histologic examination and functionally by the fact that the tone of the sphincter muscle and in some cases also the pupillary light reflex were preserved and conjugate horizontal deviation of the eyeballs could be elicited on cortical stimulation in normal extent. The nucleus of the fourth cranial nerve was outside

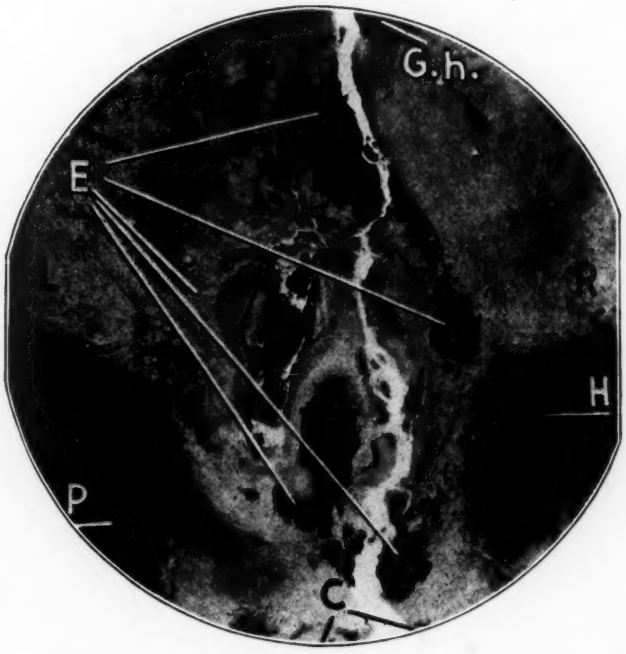

Fig. 3 (cat 14).—*E*, electrolytic lesion and hemorrhages in the central gray matter around the third ventricle, reaching from the ganglion habenulae (*G. h.*) close to the corpus mammillare (*C*). *H* indicates Forel's field H; *P*, the cerebral peduncle; *L*, the left side, and *R*, the right side.

the level of the lesions. It is, of course, possible that cells of the reticulate substance of the mesencephalic tegmentum are intercalated in parts of the pathways of the corticofugal impulses for conjugate vertical movements before these impulses reach the region of the central gray matter. The direct connection between the reticulate substance of the tegmentum and the nuclei for the ocular muscles seems, however, to be not sufficient for the cortical innervation of conjugate vertical movements in either direction, and the impulses for upward

as well as for downward movement seem to take their way over the central gray matter or closely adjacent systems before reaching the nuclei for the ocular muscles (in regard to the innervation of horizontal ocular movements see Spiegel [18]).

Small lesions of the central gray matter may induce partial loss or disturbance of vertical movements. In the following case (cat 30), for example, cortical stimulation failed to elicit upward movement of the eyeballs, although vertical deviation in either direction could be elicited by labyrinthine stimulation.

CAT 30.—On May 20, 1937, at 11 a. m. to 12, electrolysis was carried out 4 and 6 mm. anterior to the interaural line, in the midsagittal line and 1.5 mm. outside the midline on each side, the depth of puncture being 19 mm. After the lesion the eyeballs were deviated to the right, and the pupils were contracted (the transverse diameter was 2.5 mm.). There was no reaction to light (the animal was in sleep due to the injection of dial).

At 3 p. m. the animal was still in sleep induced by dial. Stimulation of the labyrinth by rotation on the Bárány chair (the head was held fixed so that the left ear was down and the right ear up) induced the following movements: After ten revolutions to the right the eyeballs deviated toward the lower lid and returned in jerky movements to the original position, and after ten revolutions to the left the eyeballs deviated toward the upper lid, then moved in opposite directions.

After exposure of the cerebral cortex on each side the eyeballs showed conjugate deviation to the left.

After stimulation of the left frontal lobe one repeatedly observed deviation of the eyeballs to the right. In one experiment this movement was combined with deviation of the head to the right; in one instance it was combined with downward movement of the eyeballs, and in another instance with convergent movement.

Stimulation of the right frontal lobe induced deviation of the eyeballs to the right or horizontal undulation combined with, or preceded by, downward movement.

Stimulation of the left occipital lobe induced only horizontal movements of the eyeballs to the opposite side.

Stimulation of the right occipital lobe induced slight horizontal undulation of the eyeballs.

Histologic Examination.—The electrode had penetrated the medial part of each anterior quadrigeminal body and produced a lesion in the dorsal part of the central gray matter (fig. 4), leaving the oculomotor nuclei and nerves intact. In the pretectal region the lesion encroached on the posterior commissure.

There was loss of the downward movement of the eyes in cat 13; the lesion was closely cranialward from the right arm of the posterior commissure and encroached on its anterior fibers. In cat 12 there was only minimal downward movement of the left eye, and distinct downward movement of the head was preserved, while upward movements of

18. Spiegel, E.: Rôle of Vestibular Nuclei in Cortical Innervation of Eye Muscles, Arch. Neurol. & Psychiat. **29**:1084 (May) 1933.

the eyes were repeatedly obtained. The lesion was a puncture in the region of the left ganglion habenulae. In cat 17 only impulses originating in the occipital lobe failed to produce vertical movements, while stimulation of the frontal lobe still elicited vertical movements in both directions, mostly downward but occasionally upward. The lesion was a puncture through the medial part of the right anterior quadrigeminal body, passing alongside the right portion of the circumference of the aqueduct and encroaching on the most dorsal part of the reticulate substance outside the right posterior longitudinal fasciculus. In cat 7 the downward movements were preserved, but instead of the conjugate

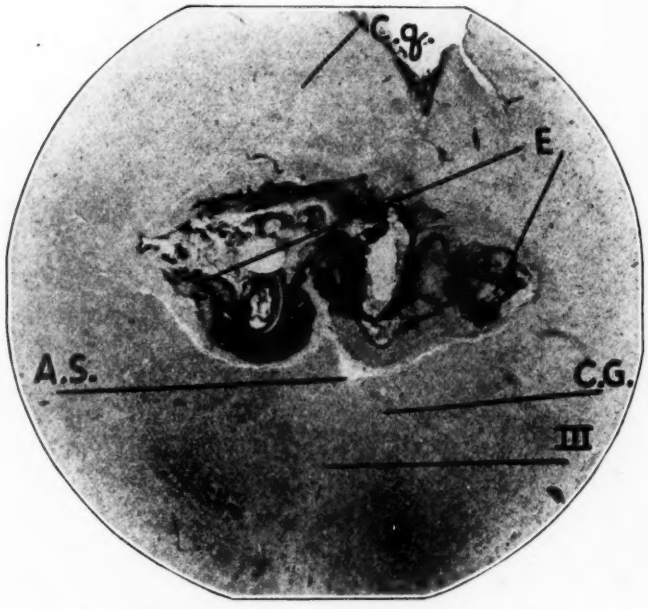

Fig. 4 (cat 30).—Electrolytic lesion (*E*) in the dorsal part of the central gray matter (*C. G.*) around the aquaeductus Sylvii (*A. S.*). *C. q.* indicates the corpus quadrigeminum, and *III*, the oculomotor nucleus.

upward movements stimulation of either frontal lobe produced after the lesion in the mesencephalic gray matter skew deviation (upward movement of the right eye and downward movement of the left eye). The lesions were bilateral punctures of the central gray matter behind the posterior commissure, more extensive on the right and encroaching on the most dorsal part of the tegmentum. Since we observed occasionally skew deviation in former experiments [19] on stimulation of the posterior

19. Scala, N. P., and Spiegel, E. A.: Effect of Stimulation of Posterior Longitudinal Fasciculus on Ocular Muscles, Arch. Ophth. **9**:939(June) 1933.

longitudinal fasciculus in the pons, it seems that this symptom may appear in diseases of various parts of the pathways innervating ocular movements and has, therefore, only limited value for local diagnosis.

In view of the fact that circumscribed lesions of the central gray matter in the cranial part of the mesencephalon may abolish the conjugate vertical ocular movements on cortical stimulation, the problem presented itself whether the pathways for this innervation reach the central gray matter by way of the tectum, which was considered by numerous authors since Adamük [20] as a coordinating center for associated ocular movements. Bernheimer [21] and Bechterew [22] made contradictory statements. In other words, the question arose whether paralysis of vertical ocular movements in diseases of the dorsal part of the midbrain should be considered as a local symptom of a lesion of the central gray matter and its adjacent nuclei, including the tectum or excluding this ganglion.

Thus, it was necessary to ascertain in further experiments which ocular movements can still be observed on cortical stimulation of the frontal as well as of the occipital lobes after destruction of the tectum of the anterior quadrigeminal bodies. Since extensive lesions had to be produced, the stereotaxic instrument seemed less fit for these experiments. After exposure of the occipital lobes they were, therefore, gradually pushed outward by introducing small pledgets of cotton between them and the falx cerebelli and the tentorium, respectively. The sagittal sinus was ligated and cut between the ligatures, and the posterior part of the falx cerebelli was excised so that the tentorium was exposed. The vena magna Galeni and the sinus rectus were coagulated; the dura covering the tentorium was removed, and the anterior part of the tentorium was excised so that the anterior lobe of the cerebellum appeared. Then the posterior quadrigeminal bodies were gradually separated from the anterior lobe of the cerebellum so that the entrance to the aqueduct became visible. A small bent knife was thrust into the midbrain below the posterior quadrigeminal bodies, parallel to the aqueduct, separating the tectum of the posterior, and then that of the anterior, quadrigeminal bodies from the central gray matter. In some experiments the tectum of the corpora quadrigemina was destroyed by electrocautery. These operations were followed by the usual stimulation experiments on the cerebral cortex, of which the following is an example.

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20. Adamük, E.: Centralbl. f. d. med. Wissensch. **8**:65, 1870.

21. Bernheimer, S.: Wien. klin. Wchnschr. **12**:1310, 1899.

22. Bechterew, W.: Funktionen der Nervenzentren, Jena, Gustav Fischer, 1909, vol. 2, p. 1041.

EXPERIMENTS WITH DESTRUCTION OF THE TECTUM OF THE CORPORA QUADRIGEMINA

CAT 23.—On April 14 destruction of the tectum of the anterior corpora quadrigemina by electrocautery was carried out. After this operation the right eye deviated downward, and the left eye deviated inward. The transverse diameter of the left pupil was 2.5 mm., and that of the right pupil was 3 mm.

The results of stimulation of the cerebral cortex are shown in protocol 2.

Histologic Examination.—The destruction of the tectum of the anterior quadrigeminal bodies by the electrocautery reached to the posterior commissure

PROTOCOL 2.—*Cat 23*

Time	Stimulated Area	Ocular Reactions	Other Reactions
11:43	Left frontal lobe..... (distance of coils, 6 cm.)	Right eye deviated to right and upward; left eye showed no movement	
11:44	Right frontal lobe.....	Each eye deviated first to left and then downward	Head deviated to left; cloni of left foreleg
11:45		Spontaneous conjugate deviation to left	
	Right frontal lobe.....	Brief movement of each eye to left	
11:46	Right and left occipital lobes	Are unexcitable	
	Right frontal lobe.....	Conjugate deviation to left	
11:47	Left frontal lobe.....	Each eye deviated to right and upward	
11:48		Each eye deviated inward and rotated	
11:51	Left frontal lobe.....	Right eye rotated outward; left eye rotated inward and deviated upward	
11:52	Right frontal lobe.....	Deviation of each eye downward and to left; pupils dilated	General cloni
	Right frontal lobe.....	Each eye deviated upward and to left; after stimulation ceased they moved downward	
11:53	Right and left occipital lobes	No effect	
11:54	Right frontal lobe.....	Each eye deviated upward and to left followed by vertical nystagmus; finally slow downward deviation	General cloni
11:55	Right frontal lobe.....	Each eye deviated downward and to left; then horizontal undulation while the eyes deviated downward	
	Left frontal lobe.....	Right eye deviated upward and to left; left eye showed rotation to left	
11:56	Right frontal lobe.....	Same reaction as at 11:55	

(fig. 5) and on more caudal sections to the dorsal portion of the circumference of the central gray matter. The lesion extended caudally to the left inferior colliculus and cranially to the medial nucleus of the right optic thalamus.

These experiments showed that stimulation of the frontal lobes may still yield vertical ocular movements in either direction after destruction of the tectum of the anterior quadrigeminal bodies. The excitability of the oculogyric centers in the occipital lobe was distinctly diminished or lost for ocular movements in either direction after such operations.

In interpreting these findings one has to bear in mind that it is necessary to push the occipital lobes lateralward in order to completely

destroy the tectal region. A certain degree of impairment of the excitability of the occipital lobes seems, therefore, hardly avoidable. In some experiments, at least, rudimentary vertical ocular movements could be elicited by stimulation of the occipital lobes after destruction of the tectum; this was taken to indicate that impulses from the occipital lobes to the subcortical mechanisms for vertical ocular movements do not take their way exclusively over the anterior quadrigeminal bodies but that conduction outside of this ganglion is also possible. This conclusion is corroborated by the fact that centrifugal pathways from the

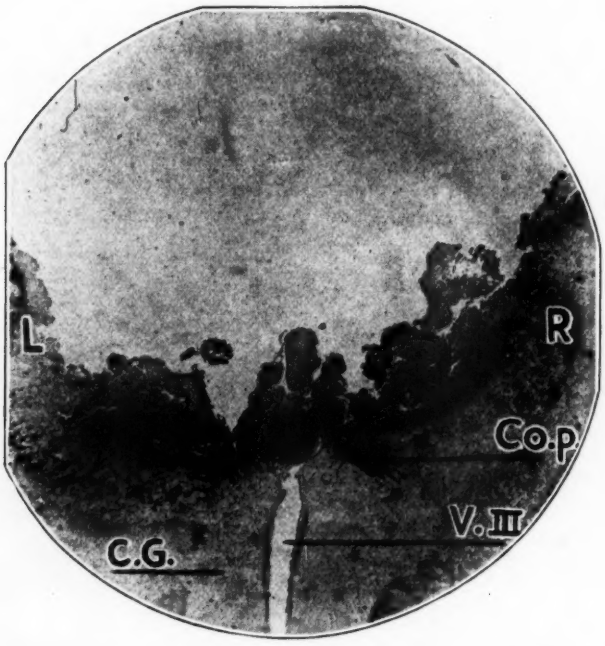

Fig. 5 (cat 23).—Destruction of the tectum of the anterior quadrigeminal body. *C. G.* indicates the central gray matter; *Co. p.*, the posterior commissure; *V. III*, the third ventricle; *L*, the left side, and *R*, the right side.

occipital lobe (the posterior lateral gyrus) could be traced by the degeneration method not only to the pretectal areas and the stratum opticum of the superior colliculus but also to the basis pedunculi, reaching the pontile nuclei (Barris [23]). As for impulses from the frontal lobe, it can be stated with certainty that they can reach the subcortical centers for upward and downward movements of the eyeballs independently of the tectum. Thus the loss of voluntary vertical ocular movements, which originate in the frontal lobe, in cases of lesions in the neighbor-

23. Barris, R. W.: J. Comp. Neurol. **63**:353, 1936.

hood of the aqueduct of Sylvius cannot be ascribed to the destruction of the systems taking their way over the tectum.

The question as to which special group of cells in the region of the central gray matter or its immediate vicinity is concerned in the corticofugal impulses for vertical ocular movements to the nuclei for the ocular muscles requires further studies. Some of our observations direct attention to the nuclei in the region of the posterior commissure, the nucleus of Darkschewitsch and particularly the nucleus interstitialis of Cajal, where descending fibers of the posterior longitudinal fasciculus originate (Muskens [24] and van der Schueren [25]). We mentioned the nearness of the stimulation points to the light reflex arc of the pupil and the fact that downward movements were elicited with threshold currents by stimulation closely dorsal to the oculomotor nucleus. The characteristics would correspond to the location of the nuclei of the posterior commissure. Further experiments, however, seem necessary to show on which groups of cells the fibers that carry corticofugal impulses for vertical ocular movements synapse. As for the pathways by which these corticofugal impulses reach the region of the mesencephalic central gray matter, this much can be said: The globus pallidus and its efferent systems are dispensable for such conduction, since we obtained vertical ocular movements in either direction on cortical stimulation after transection of the pallidofugal fibers.[9]

SUMMARY

1. Stimulation of the central gray matter around the aqueduct of Sylvius below the cranial part of the anterior quadrigeminal body may yield constriction or dilatation of the pupil according to whether the pathway of the light reflex or the descending vegetative tracts originating in the hypothalamus are stimulated. Lesions of the central gray matter in this level produce, at most, transitory disturbances of the light reflex; usually they have a short-lived stimulating effect on the sphincter of the pupil, which shows a tonic type of reaction—retardation of its relaxation after cessation of constrictor impulses or on application of dilator impulses.

2. One obtains conjugate downward movements of the eyeballs by stimulation of the central gray matter below the posterior commissure or behind this level and upward movements by stimulation in the posterior part of the optic thalamus or in the medial part of the tectum of the anterior quadrigeminal body. The downward movement is often

24. Muskens, L. J. J.: Brain **36**:352, 1914.

25. van der Schueren: Névraxe **13**:183, 1912.

associated with downward movement of the head in the same direction. The system innervating downward movements lies in the vicinity of the pupillary light reflex arc and the descending hypothalamic pupillodilator tracts; it lies close and dorsal to the stimulation point for convergent movements. Destruction of the central gray matter in the cranial part of the mesencephalon and the pretectal region induces loss of upward and downward movements on cortical stimulation. A small lesion of this region may be followed by various partial disturbances of vertical ocular movements (loss of conjugate movements in one direction only, the appearance of skew deviation on cortical stimulation or loss of vertical movements on stimulation of certain cortical areas, e. g., the occipital lobes only).

3. The tectum mesencephali is not an indispensable part of the system which transmits impulses for vertical ocular movements from the frontal lobes to the nuclei for the ocular muscles. Paralysis of vertical conjugate movements in diseases of the dorsal part of the mesencephalon with intact nuclei for the ocular muscles should therefore be regarded as a local symptom of a lesion of the region around the aqueduct of Sylvius or of closely adjoining systems (the nuclei in the region of the posterior commissure?) but not as a local symptom of a lesion of the tectum.

ABSTRACT OF DISCUSSION

Dr. Clifford B. Walker, Los Angeles: The ophthalmologist as well as the neurosurgeon appreciates most intensely the work of Dr. Spiegel and Dr. Scala on the central connections and pathways of the ocular nerves. In this difficult and intricate field of investigation the findings of Dr. Spiegel and his school deserve the greatest praise. Dr. Spiegel's opening remark, "The region around the aqueduct of Sylvius is of special interest from an ophthalmologic point of view," is certainly true.

Among the numerous ocular disturbances that may come from injuries in the mesencephalic region to the nuclei or pathway of certain nerves, particularly those having control of the oblique muscles, I believe it is possible to include a common type of separated retina.

The following facts and arguments may be considered in this connection:

1. Retinal tears occur so commonly in the upper temporal quadrant in the neighborhood of the attachment of the superior oblique muscle that this is regularly the first region to be investigated by me in routine examination.

2. In over two hundred cases separation of the retina has occurred in only one eye at the same time and from the same trauma. Although double or simultaneous separation does occur, it is decidedly uncommon.

3. The upper portion of the retina is phylogenically older and stronger than the lower portion, yet it is at a mechanical disadvantage

as compared with the lower portion by at least inertia due to the weight of the vitreous (which may weigh over 7 Gm. in myopic eyes). This is because the uppermost portion of the retina, especially if the eye has become softened, must contend with the sag of the vitreous, which tends to produce an area of low pressure between the upper portion of the vitreous and the sclera, while below the vitreous is pressing the retina to the choroid by its weight, causing an area of higher pressure.

4. The superior oblique muscle is attached in the area of low pressure at the top of the eye at the point of termination of the ocular motion of looking downward and outward, and the tendon of this muscle pulls almost at right angles to this spot on the globe on one eye at a time—the right eye, for instance, in looking downward and to the right. A right-handed person has his eyes in this position in many instances at the moment of an accidental jolt that brings on separation. On the other hand, if a person (an actual case is considered) sitting on the right side of an automobile is thrown to the roof by a dip in the road, he very likely will be looking down and to the left for the best place to land. On the way up probably he is looking for a spot without handles and glass on which to fall. He looks down to his left. This direction of gaze may be present on the way down also (if he is not too dazed by bumping the head above) until the spine and pelvis rebound on the seat. My theory is that the fourth nerves receive then a special stimulus which is either direct or transmitted to the mesencephalic decussation in the roof of the fourth ventricle or the Sylvian aqueduct. This extra stimulus may affect each fourth nerve alike, but only one superior oblique muscle—in this case the right superior oblique muscle is already tensely pulling at an angle of about 60 degrees and elevating the sclera when the extra stimulus comes. The other superior oblique muscle is at the same time relaxed. The superior oblique muscle, the longest (60 mm.) ocular muscle, is able to act in this manner because it still has contracting ability to jerk the sclera while the eyeball is held in the position of maximum disadvantage, since its long (20 mm.) tendon is then shortened only about one-half (in the pretrochlear portion) as its attachment comes more directly under the pulley. The shorter the contact arc (5 mm. or less) of the superior oblique muscle (the sagittal type of Fuchs) the more pronounced this action may be. Also, the softer and more weighty the eye (as in myopia) the more effectively can the superior oblique muscle act in the manner just described. The other ocular muscles do not possess to the same extent this mechanical disadvantage making them liable to injury.

It is in the dorsal portion of the mesencephalon (in the wall of the aqueduct, extending to the fourth ventricle), where the fourth nerve decussates more fully than does the optic nerve anteriorly, that it is in a position to receive a stimulus from end thrust impact along the spinal column. From this decussation the fourth nerve winds a long course around both the cerebral and the cerebellar peduncle to reach the orbital fissure. A longitudinal spinal impact may be given lateral components by these peduncles so as to jerk on the anchorage produced by the decussation, which is embedded in a fluid retaining wall somewhat analogous to the chiasmal arrangement of the optic nerve under the

third ventricle anteriorly. Not only can the chiasm be stimulated by certain impacts but it can even be torn sagittally by them.

Dr. W. Lancaster, Boston: I should like to ask Dr. Walker what makes him think that the vitreous weighs 7 Gm.

Dr. Clifford B. Walker, Los Angeles: That is an approximate estimate. When I say that the disadvantage due to weight is 7 Gm. I mean that the weight on the lower portion of the retina is helpfully pressing, while that on the upper portion of the retina is not so helpful and by its inertia contributes to the pressure on the lower portion in case of an upward jerk on the sclera.

Dr. Walter Lancaster, Boston: The whole eyeball is not supposed to weigh 7 Gm.

Dr. Clifford B. Walker, Los Angeles: Its weight has been estimated as between 7 and 8 Gm. If the sclera is removed the weight will be less. Yet so many of the patients with this condition are myopic that some allowance toward the larger number seems fair. But if one fills this ball with water one will find that a sphere 24 mm. in diameter has a weight of about 7 Gm., and the eye has a specific gravity a little over unity.

Dr. Norman P. Scala, Washington, D. C.: I do not know that I can add any more to what has already been said. I wish, however, to thank Dr. Walker for his generous discussion of the paper. Dr. Spiegel and I were of the opinion that supranuclear centers existed for vertical conjugate ocular movements, and we think we have proved that such centers do exist. Paralysis of voluntary vertical conjugate ocular movements should not be considered as a local symptom of a lesion of the tectum mesencephali but as a local symptom of a lesion of the region around the aqueduct of Sylvius or some neighboring or adjoining systems, probably nuclei in the region of the posterior commissure.

Ophthalmologic Review

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ORBITAL ABSCESSES

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Orbital abscess is frequently used in its broadest sense to include all types of acute inflammation of the walls and soft tissues of the orbit. Such usage has the virtue of brevity and is perhaps proper in such a review as this. However, in actual clinical work it is of the greatest practical importance to differentiate between a true abscess in the retrobulbar space and a disease limited to the rim or wall of the orbit, such as periostitis, osteomyelitis or subperiosteal abscess. All these conditions may produce the characteristic signs of acute swelling of the lid, proptosis, deep orbital pain and fever. In addition, simple congestion of the orbital veins due to swelling of the mucous membrane in the ethmoid sinuses, as well as extensive spreading thrombophlebitis of the orbital venous system, may produce the same characteristic signs.

Ophthalmologists of fifty or seventy-five years ago were aware of these differences in pathologic anatomy probably just as well as are ophthalmologists of today, but their routine practice of making immediate exploratory incisions into the soft parts of the orbit indicates that they did not fully appreciate the practical importance of making these differentiations. Of course, the one thing which has been of the greatest help to the ophthalmologist in understanding orbital inflammations is the appreciation of the fact that disease of the accessory nasal sinuses is their most frequent cause. Stellwag von Carion [1] in 1868 made no mention whatever of sinus disease in this regard. However, many of the early writers mentioned exposure to cold as a cause of orbital abscess. Fuchs [2] in 1893 stated that retrobulbar phlegmon may arise from periostitis of the wall of the orbit adjacent to an infected nasal sinus. During recent years the sinusal origin of orbital infections has become more and more appreciated. Birch-Hirschfeld [3] expressed the opinion

1. Stellwag von Carion, C.: Treatise on Diseases of the Eye, translated and edited by C. E. Hackley and D. B. St. John Roosa, Baltimore, William Wood & Company, 1868.

2. Fuchs, E.: Textbook of Ophthalmology, ed. 2, translated by A. Duane, New York, D. Appleton & Company, 1892, p. 584.

3. Birch-Hirschfeld: Clinical Course and Therapy of Orbital Phlegmons, Auerbach Jubilee Volume, 1935, p. 84.

that in 60 per cent of cases orbital cellulitis is caused by disease of the nasal sinuses. In his series of six hundred and eighty-four cases in which it was due to sinus disease, the frontal sinus was diseased in 28.8 per cent, the antrum in 21.8 per cent, the ethmoid sinus in 20.5 per cent and the sphenoid sinus in 6.1 per cent. Several sinuses were affected in 14.7 per cent. In a recent report he condemned exploratory puncture into the soft parts of the orbit because it may carry infection into uninvolved tissue and advised making an incision down to the rim of the orbit, followed by elevation of the periosteum until the site of the infection is reached. These are truly words of wisdom from one who has studied the problem for many years. This type of surgical treatment is indicated chiefly in the cases of orbital inflammation due to sinus disease and only in certain stages in such cases. There can be, of course, no routine treatment applicable in all cases at any stage of the development of the inflammation.

ETIOLOGY

The etiologic factors of orbital inflammation are as follows:

1. Extension of infection from adjacent tissues, such as the accessory nasal sinuses, the teeth and the lacrimal sac, and infections of the lid, including erysipelas.

2. Trauma—penetrating and nonpenetrating injuries, including operations on adjacent structures.

3. Bacteremia.

The importance of the nasal sinuses in orbital inflammation is obvious when one considers the frequency with which they become diseased and, further, considers the fact that only a thin layer of bone and two layers of periosteum separate the sinuses from the orbit. There may even be dehiscences in the bone, and arteries, veins and nerves extend through these separating partitions. There is as yet no proof that the lymphatics of the sinuses communicate with those of the orbit, but they may do so. The superior ophthalmic vein communicates with the ethmoid vein; occasionally the inferior ophthalmic vein does likewise. Batson [4] stated that he did not consider the arteries or lymphatics to be important channels for the spread of infection from the nose to the orbit, but that he did consider the veins to be the important route. He also found that when an opaque substance was injected into the anterior facial vein it spread to the superior ophthalmic vein and many of its tributaries. Martin Cohen [5] also stated the belief that the veins are the

4. Batson, O. V.: Relationship of the Eye to the Paranasal Sinuses, Arch. Ophth. **16**:322 (Aug.) 1936.

5. Cohen, Martin: Inflammatory Exophthalmos in Catarrhal Disorders of the Accessory Sinuses, Tr. Sect. Ophth., A. M. A., 1935, p. 209.

important factor. The periorbita is rather loosely attached to the bone except around the rim of the orbit, at the apex and along the various sutures. It can therefore be easily elevated from the bone by collection of pus, with the production of a subperiosteal abscess.

The ethmoid sinus is present at birth, and infection in it can give rise to orbital inflammation at any age. This is the most frequent cause of orbital disease in infants and young children. The antrum is also present at birth but is relatively higher and more mesial than in later years. In infants it is usually diseased if the ethmoid cells are diseased and affects the orbit in much the same manner. The frontal sinuses are not developed at birth and do not become a factor in orbital disease until about the seventh or eighth year. From then on they become an exceedingly important factor. Intracranial complications occur more frequently in cases of orbital disease resulting from infection of the frontal sinus than in the cases of orbital disease due to ethmoiditis. It is probably a fact that whichever sinus is most rapidly developing at a given age is most likely to cause orbital inflammation if it becomes acutely diseased. Thus, one finds disease of the ethmoid sinus and antrum to be the common cause of orbital inflammation in infants and children up to the age of 9 or 10. Then the frontal sinus becomes more important. In adults these three sinuses are about of equal importance.

Infection in the tooth buds furnishes another example of the tendency for inflammation to arise in rapidly developing tissues. This occurs most often in nursing infants and may arise as a result of trauma to the mucous membrane of the gums, the infection spreading upward into the tooth buds and then to the floor of the orbit, as has been suggested by Marx.[6] A different origin for this type of infection was suggested by van Gilse,[7] who stated that the bony case of the maxillary sinus is not completely closed in an infant, so that infection may pass from the nose to the antrum and then to the tooth buds, with resulting osteomyelitis of the superior maxilla.

Infection in the lacrimal sac may spread to the orbit either spontaneously or as a result of operations. This happens less often than one would expect, probably because the deep fascia covering the sac gives excellent protection. I have seen it occur as a result of too liberal use of trichloro-acetic acid and also following curettage of the interior of the sac. John Green[8] has reported a case of orbital abscess which followed hordeolum.

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6. Marx, E.: Eye Symptoms Due to Osteomyelitis of Superior Maxilla in Infants, Brit. J. Ophth. **6**:25 (Jan.) 1922.

7. van Gilse, P. H.: Die pathologische Bedeutung der Nebenhohlen der Nase bei kleiner Kinder, Maandschr. v. kindergeneesk. **3**:431, 1934.

8. Green, John: Management of Orbital Infections, Am. J. Ophth. **14**:196 (March) 1931.

Erysipelas must have been a common cause of orbital disease in years past, judging from the prominent place given to it by the early writers. One early textbook makes the statement that in many cases of orbital cellulitis of apparently unknown origin the condition was probably due to slight attacks of erysipelas of which the patient was not aware. The physicians of those days must have been erysipelas conscious as a result of their experiences in military surgery. Certainly it is not a common cause of orbital disease at the present time. However, Bellows [9] found that among one hundred and thirty patients with facial erysipelas, six had orbital cellulitis. He expressed the belief that it develops by thrombophlebitis and leads to multiple small abscesses which may become confluent. Incision may be inadequate, and in one case evisceration of the orbit was done because there were threatening signs of meningitis.

Trauma may cause orbital inflammation without any penetrating injury being present. This ordinarily occurs as a contusion from injury from a blunt instrument and results in periostitis of the rim of the orbit. However, this may spread deeper to the wall of the orbit and cause proptosis. Penetrating injuries from foreign bodies of various types are more common and are more serious. Organic materials, such as splinters of wood, often harbor bacteria and are therefore more prone to cause suppuration than glass or metallic substances. Surgical traumas, such as the extraction of infected teeth and operations on the nasal sinuses, at times result in malignant orbital infection, frequently of a thrombophlebitic type.

Bacteremia causes a considerable proportion of orbital infections. As a rule, the process begins as periostitis, but because the patient is already seriously ill it rapidly spreads into the retrobulbar tissues and forms a true orbital abscess. This occurs most frequently as a complication in the exanthematous diseases, especially scarlet fever and smallpox and, to a lesser extent, measles. It occurs less often in pneumonia and puerperal sepsis. In the exanthematous diseases it is often difficult to determine whether the orbital disease is due to bacteremia or is a result of the purulent rhinitis which often is present. In cases of orbital abscess complicating scarlet fever the infective agent is ordinarily the streptococcus; in cases of smallpox and measles the orbital abscess is due to secondary invaders. In general, the streptococcus, the hemolytic staphylococcus and the pneumococcus are the organisms found in the abscess. Beigelman [10] reported the case of a patient who had an actino-

9. Bellows, John: Ocular Complications of Erysipelas, Arch. Ophth. **11**: 678 (April) 1934.

10. Beigelman, M.: Actinomycosis of the Orbit, Arch. Ophth. **10**:664 (Nov.) 1933.

mycotic infection in the orbit. The process lasted for two years, and death occurred from low grade meningitis and abscess of the brain. Adams [11] found reports in the literature of eight cases of infections in the sinuses due to Aspergillus and added a case of his own. Eight patients were women, and only one was a man. Proptosis was an early sign, and there was a profuse nasal discharge.

PATHOLOGICO-ANATOMIC FEATURES

It is not always possible to determine the pathologico-anatomic features from the clinical appearance, but careful consideration of the etiology will greatly aid one's judgment in this respect. When disease of the nasal sinuses is the cause, the nasal wall of the orbit is usually involved. In children this is true in regard to the frontal and maxillary sinuses, as well as the ethmoid sinus, because they have not extended laterally very far. In older patients the upper and lower quadrants are affected in disease of the frontal and the maxillary sinuses, respectively. In many of the cases of milder inflammation there is only venous congestion in the orbit for a few days, due to compression of the ethmoid vein by the swollen mucous membrane in the ethmoid sinus. Edema of the upper lid and slight proptosis may be the only signs. This type is common in young children. If the sinus disease becomes more extensive it may cause some osteomyelitis of the nasal wall of the orbit, and the infection may cause a subperiosteal abscess. This leads to greater swelling of the lid and greater proptosis and usually to fixation of the eyeball. This abscess may penetrate the periosteum and cause an abscess in the soft parts of the orbit, or it may extend back to the apex of the orbit and cause meningitis or thrombosis of the cavernous sinus, but it usually dissects forward and appears as a subcutaneous induration in the lids, usually above or below the internal canthal ligament. In these cases the true contents of the orbit are spared, and recovery is complete and prompt as soon as the subperiosteal abscess is drained. When such an abscess points in the conjunctiva, either the fornix or on the eyeball, it means that the pus has dissected through the soft tissues, and the orbital structures may be permanently damaged. In some cases the subperiosteal abscess drains back into the nose, and recovery is spontaneous. This is true of the cases of orbital abscess due to sinus disease, not of cases of orbital abscess of the other types.

Many times the process starts as acute periostitis. This is true especially of those cases in which the orbital abscess is due to nonpenetrating injury of the orbital rim and of those cases in which it is

11. Adams, N. F., Jr.: Infection Involving the Ethmoid, Maxillary and Sphenoid Sinuses and the Orbit Due to Aspergillus Fumigatus, Arch. Surg. **26:** 999 (June) 1933.

associated with bacteremia. In the cases in which it is due to nonpenetrating trauma it is usually not severe. In the cases in which it is due to penetration of foreign bodies it varies considerably in severity according to the type of infection carried in. Splinters of wood usually cause more severe inflammations than other injuries and, incidentally, are more likely to cause tetanus. The type of orbital abscess associated with bacteremia is very severe and unless promptly treated usually extends into the orbit and causes an abscess of the soft parts, frequently resulting in damage to the optic nerve or the nerves to the muscles.

Osteomyelitis of the superior maxilla occurs most frequently in infants and children and appears to arise spontaneously. The acute form is most common. The entire cheek is swollen and indurated and is often dark. In 10 per cent of these cases the floor of the orbit is involved sufficiently to cause proptosis. There is considerable swelling of the lid in practically all cases. The mortality in cases of this type is about 25 per cent. The entire maxilla is usually involved, and discharging fistulas may appear along the lower rim of the orbit, through the palate or into the canine fossa. Ultimately a chronic stage is reached in which the acute swelling subsides but the fistulas remain. Rough bone denuded of periosteum may be felt at the bottom of these fistulas, and spicules of loose bone may be extruded. There is also a type of osteomyelitis in which the infection is of low grade throughout its course. The fistulas appear in the lower lid and cause adherent depressed scars. There may also be discharging fistulas along the alveolar process in association with very carious teeth. This type of infection resembles the chronic inflammation of the superior maxilla due to tuberculosis or syphilis.

SIGNS

In addition to the general evidences of infection, fever and leukocytosis (which are not invariably present), the cardinal signs of orbital inflammation are redness and swelling of the lids and conjunctiva, proptosis and fixation of the eyeball. Chemosis occurs when the pus nears the surface. Induration of the subcutaneous tissues results when the abscess has dissected its way forward under the skin.

COMPLICATIONS

The question of complications resulting from orbital inflammation deserves careful consideration. In some cases immediate and radical treatment is essential; in others too much treatment is literally fatal. The important factors are the severity of the condition, the cause, the age of the patient and the status regarding other existing complications.

In young children orbital inflammation due to sinus disease is usually not very dangerous if handled in a conservative manner. Many of the

patients recover spontaneously or with simple treatment. As a rule, even though an abscess forms, it is subperiosteal and readily dissects its way forward and can be evacuated by a simple incision of the skin. The few days required for this to occur gives time for a protective pyogenic membrane to form. Complications in this type of orbital inflammation are not common. If an incision is made too early or if extensive intranasal or external operations on a sinus are done in these cases of acute disease the mortality is very high, especially in patients under 2 years of age, and death occurs more often from pneumonia, empyema and general sepsis than from meningitis or disease of the cavernous sinus. This is an important fact and is, perhaps, more thoroughly appreciated by the pediatrician than by the ophthalmologist, or the rhinologist. In children 10 years of age or more, disease of the frontal sinus is often the cause. In these patients the condition may be very severe and may frequently result in abscess of the frontal lobe of the brain or meningitis. In most cases of orbital inflammation there is probably more or less infective thrombophlebitis. In those cases in which an abscess forms in the orbital contents this condition is probably extensive. Occasionally it spreads to the cavernous sinus, but not nearly as often as one would expect. Retrograde extension of infective thrombophlebitis into the optic nerve and eyeball probably accounts for the neuritis and the later atrophy of the optic nerve which are often seen, and also for the rare occurrence of massive exudative choroiditis, which may be found associated with severe involvement. A slight congestion of the retinal veins is commonly seen and is of no great importance. Ulceration of the cornea may occur as a result of exposure resulting from the exophthalmos and is perhaps aided by a certain degree of damage to the ciliary nerves due to pressure within the orbit. Also, corneal abrasions may be caused by gauze dressings and drains. The corneas of adults are less resistant to ulceration than are those of children, but the latter are not exempt from ulceration.

Strabismus of a paralytic type sometimes follows orbital abscess, either because the nerve to a muscle has been damaged within the orbit or because the action of a certain muscle is limited by scar tissue. In children it is not unusual to have the onset of impending concomitant squint precipitated simply because for several weeks the eye has been occluded by a dressing or swelling of the lid, or both. It is wise, therefore, in all cases of orbital abscess in children to occlude the unaffected eye for certain periods after recovery, to prevent suppression of vision.

TREATMENT

The treatment of acute inflammations in the orbit is to a great extent determined by what one knows concerning the etiology and the pathologico-anatomic features. Immediate routine exploratory incision

through the conjunctival fornix into the soft retrobulbar tissues in all cases of acute proptosis is a thing of the past. There are several reasons for condemning this practice: There may be no pus to evacuate; the pus may be entirely subperiosteal (and in that event the infection would gain access to the retrobulbar tissues as a result of the operation), and in many cases it is safer to allow time for the abscess to become walled off. On the other hand, the danger of permitting a true orbital abscess to continue undrained is considerable. Mention has previously been made of the fact that Birch-Hirschfeld condemned exploratory puncture and advocated a subperiosteal approach. Hall [12] reported twenty-three cases of orbital infection in which there were four deaths. He divided his cases into two groups, thoses in which the condition was due to external infection such as furuncles, erysipelas and trauma and those in which it was due to sinus infection. All the deaths occurred in the first group. The patients required radical treatment. He expressed the belief that patients in the second group should have more conservative treatment. A number of writers have pointed out that in a high percentage of cases of orbital infection due to sinus disease the patient recovers safely following the application of astringents to the nasal mucous membrane and nasal suction or, at most, incision through the skin to evacuate a subperiosteal abscess after it has neared the surface. Among these writers are Moller,[13] Ballenger,[14] Gamble [15] and Greenfield.[16] Greenfield in cases of orbital infection due to sinus disease uses nasal astringents and suction, and performs incision and drainage only if the eye becomes fixed and induration is present. In two cases he was able to demonstrate by roentgen examination that the ethmoid labyrinth was dilated; that is, the distance between the nasal septum and the orbital plate was greater on the affected side than it was on the normal side.

The results obtained by those advocating this type of treatment have been good. A number of other writers who have favored external operations on the sinuses in similar cases, such as Porter [17] and Phelps,[18]

12. Hall, J. S.: Orbital Infections, J. Laryng. & Otol. **50**:690 (Sept.) 1935.

13. Moller, J.: Ueber orbital Phlegmone, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **25**:458, 1935.

14. Ballenger, H. C.: Orbital Cellulitis and Abscess Secondary to Sinusitis, Illinois M. J. **61**:128 (Feb.) 1932.

15. Gamble, R. C.: Acute Inflammations of the Orbit in Children, Arch. Ophth. **10**:483 (Oct.) 1933.

16. Greenfield, S. D.: Acute Sinusitis in Children Associated with Orbital Complications: The Conservative Treatment, Laryngoscope **44**:683 (Sept.) 1934.

17. Porter, C. T.: The Etiology and Treatment of Orbital Infections, Ann. Otol., Rhin. & Laryng. **41**:1136 (Dec.) 1932.

18. Phelps, K. A.: Cellulitis of Orbit in Infants and Children, Ann. Otol., Rhin. & Laryng. **33**:1391 (Dec.) 1924.

have reported results which are perhaps equally good. It may be said, however, that this type of treatment does not yield as good results in young children as it does in older patients.

There have been a number of cases of exceptionally severe orbital infection which has required still more radical surgery. Eagleton [19] described infections in the retrobulbar space of thrombophlebitic nature which led to sudden blindness from arterial spasm and were part of the picture of thrombosis of the cavernous sinus. He advised immediate drainage of such abscesses by a latero-orbitocranial approach which involves rather extensive removal of bone. Martin Cohen [5] performed a Krönlein operation in two cases of long-lasting severe exophthalmos due to chronic sinus disease, and in one case he performed a Naffziger operation.

In regard to osteomyelitis of the superior maxilla, all writers have emphasized the serious nature of the disease and the difficulties in treating it. Posey [20] mentioned the need for multiple operations, including incision for drainage and later removal of sequestrated bone and dead teeth. Custodis [21] advised early and extensive surgical treatment. He reported 5 cases; 4 of the patients died. Marx [6] refrained from even making an incision for drainage in this type of case. The process is so diffuse and the induration so marked that even multiple incisions do not give adequate drainage. Pneumonia or general sepsis frequently follows such surgical intervention.

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19. Eagleton, W. P.: Exophthalmos from Surgical Diseases, Arch. Ophth. **14**:1 (July) 1935.

20. Posey, W. C.: Orbital Cellulitis from Disease of the Superior Maxilla in Children, Tr. Sect. Ophth., A. M. A., 1912, p. 229.

21. Custodis, E.: Inflammatory Diseases of the Orbit in Osteomyelitis of the Superior Maxilla in Infants, Klin. Monatsbl. f. Augenh. **87**:631 (Nov.) 1931.

Correspondence

AN ANACHRONISTIC STATEMENT

To the Editor:—In a paper entitled "Secondary Cataract with Particular Reference to Transparent Globular Bodies," by Drs. Alfred Cowan and Wilfred E. Fry, published in the July issue (ARCH. OPHTH. **18:** 12, 1937), on page 22 there is the following statement referring to a paper by Elschnig: "In the report of neither case is there an accompanying description of the results of clinical or slit lamp examination." On page 12 the reference to Elschnig's paper is given (*Klin. Monatsbl. f. Augenh.* **49:** 444, 1911).

It seems to me that the statement is rather anachronistic, considering the fact that Alvar Gullstrand published his illumination method for the first time in 1911 (Demonstration of Nernstslitlamp, Ber. ü. d. Versamml. d. ophth. gesellsch. **37:** 374, 1911), and owing to the efforts of O. Henker, of the Zeiss Works, the Gullstrand lamp was combined with the corneal microscope in 1916. So far as I remember, the pioneer publications date from 1918, and therefore Elschnig could not have given a description of the results of slit lamp examination in 1911.

ANDREW RADOS, M.D., Newark, N. J.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

National Society for the Prevention of Blindness.—The annual conference of the National Society for the Prevention of Blindness was held in New York on October 6 to 8. This year the opportunity was given to participate in the American Public Health Association's annual convention. Discussions and papers were presented on the following subjects:

"Saving Sight Through Public Action," by Dr. William F. Snow.

"Syphilis of the Eye as a Factor in Industry," by Dr. Park Lewis.

"Possibilities for Further State-Wide Development of Sight Conservation Through Cooperation of Interested Agencies" (informal discussion).

"The Vision of the School Child," by Francia Baird Crocker, R. N.

International Organization Against Trachoma.—There will be sessions of the International Organization Against Trachoma during the period of the Fifteenth International Congress of Ophthalmology, to be held in Cairo, Egypt, from Dec. 8 to 14, 1937. On December 9 there will be a meeting of the present executive committee, which will be followed by a plenary meeting of the delegates from the national ophthalmologic societies together with the subscribing members of the International Organization Against Trachoma. The names of such delegates should be sent to the president of the International Organization Against Trachoma, 33 Welbeck Street, London, W. I., at least thirty days before the opening of the congress. The following program of the scientific sessions will be presented:

Introductory address by the president, Dr. MacCallan.

"The Microbiologic Etiology of Trachoma," by Dr. Phillips Thygeson, United States of America; Dr. Grüter, Germany; Dr. Oguchi, Japan; Drs. Cuénod and Nataf, Tunis; Dr. Poleff, Morocco, and Dr. Rötth, Hungary.

"The Pathology of Trachoma," by Dr. Wilson, Egypt; Dr. Michail, Roumania; Dr. Mulock-Houwer, Netherland Indies, and Dr. Pascheff, Bulgaria.

"The Treatment of Trachoma," by Dr. Sobhy Bey, Egypt, and Dr. Shimkin, Palestine.

"The Epidemiology of Trachoma in Japan," by Dr. Motegi, Japan.

"Trachoma Among School Children in Buenos Aires," by Dr. Lijó Pavía, Argentina.

"Trachoma in Turkey," by Dr. Ayberk, Turkey.

UNIVERSITY NEWS

Dr. Francis P. Guida, for the past two years resident in ophthalmology at Yale University, has been appointed full time instructor in ophthalmology at the University of Chicago.

Dr. Louis Bothman has been promoted to the position of clinical professor of ophthalmology at the University of Chicago.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

RELATION BETWEEN THE MUCOPROTEIN AND THE FRAMEWORK OF THE VITREOUS. J. GOEDBLOED, Arch. f. Ophth. **137:** 131 (April) 1937.

A comparison of the properties of the whole vitreous with those of the dialyzed fluid of the vitreous reveals a striking congruity. The properties of the dialyzed fluid of the vitreous are determined by the specific protein present in that fluid, namely, mucoprotein. Goedbloed therefore assumes that the actual structure of the vitreous is that of filaments made up of the residual protein (vitrein) surrounded by films of mucoprotein. Such a conception would explain why, colloidochemically, the whole vitreous behaves like mucoprotein.

P. C. KRONFELD.

CYTOLOGIC CHARACTERISTICS OF THE HUMAN RETINA. Y. UYAMA, Arch. f. Ophth. **137:** 318 (June) 1937.

The horizontal cells are better developed in the human retina than in the retina of the other mammals. Neurofibrillae form a network which connects the horizontal cells directly with one another, without the action of their processes. The crystalloid bodies which Kolmer described in the horizontal cells are probably cholesterol crystals and have nothing to do with the mitochondria or other protoplasmic parts of the cell body. Of the amacrine cells two types can be distinguished, namely, spherical or elliptic cells with long processes, and smaller, but more numerous cells which contain neurofibrillae only on one side of the nucleus. These fibrillae describe conspicuous spirals before emerging from the cell bodies.

P. C. KRONFELD.

Biochemistry

OBSERVATIONS ON THE REDUCING SUBSTANCES (GLUCOSE) OF THE AQUEOUS AND VITREOUS HUMORS OF THE EYE. W. M. JAMES and A. J. SIEFKER, Am. J. Ophth. **19:** 975 (Nov.) 1936.

After carrying out experiments on rabbits, James and Siefker arrived at the following conclusions:

"1. In fasting rabbits under dial-Ciba anesthesia the blood sugar averages 137 mg. percent, the aqueous sugar 164 mg. percent, and the vitreous sugar 104 mg. percent.

"2. Changes in the glucose level of the blood are reflected after a lapse of time in the intraocular fluids. An increase in the glucose content of the aqueous increases the osmotic pressure in the anterior chamber, and with the subsequent fall of the blood-sugar level the accumulation of glucose in the aqueous causes the inflow of fluid into the anterior chamber with a subsequent rise in intraocular pressure.

The relatively lower osmotic pressure of the vitreous humor facilitates the passage of fluid from the posterior to the anterior segment of the eye.

"3. Eserine increases the rate of the diffusion of glucose into the aqueous of the eye.

"4. Atropine retards the diffusion of glucose into the aqueous humor of the eye.

"5. Irritation of the vascular bed of the uveal tract is associated with wide fluctuations in intraocular pressure. These fluctuations are due to vascular dilatation or constriction and also to alterations of the permeability of the capillary membrane."

W. S. REESE.

RESEARCHES CONCERNING THE CHEMICAL CONSTITUENTS OF THE RETINA, THEIR RELATIONS TO THE RETINAL PURPLE AND THE THEORY OF THE DUALITY OF VISION. M. VERRIER and R. PANNIER, Bull. Soc. d'opht. de Paris, July 1936, p. 569.

The theory of the duality of vision has for a long time dominated the concept of the physiologic nature of vision. All the recent research seems to rest on a small number of morphologic facts, not generally authenticated, while the facts contrary to the theory have been neglected. Instead of the dual morphologic nature of the visual cells it is convenient to substitute a notion of a polymorphic assembly of these elements. This polymorphism is found by a comparative study of the retina of man and of the vertebrates. The authors have found that the reparation of the visual purple is independent of the form of the visual cells. A chemical study of the visual cells would apparently bring forth many facts of interest concerning the matter. Wald's work, which identified the retinal purple as vitamin A, is most important. This work was done on the frog and some of the fishes. Brunner, Baroni and Kleinau reported the coexistence of B carotene and the rods, but they limited their study to the eyes of beef and swine without establishing a comparison with the retinas unprovided with typical rods. Finally and more recently, Euler, Hellström and Adler placed the localization of the flavine in the retina at the level of epithelial pigment. Their observations were made on the rabbit, the rat, the ox and man.

In the present article the authors report studies on solution of taurocholate, vitamin A, carotene albumin, melanin, flavine and other substances. They conclude that the retinas of various species contain the same constituents in varying proportions.

L. L. MAYER.

Cornea and Sclera

KERATOCONJUNCTIVITIS WITH ADENITIS IN CALCUTTA. S. SANYAL, Am. J. Ophth. **19:** 982 (Nov.) 1936.

A form of keratoconjunctivitis is described, characterized chiefly by small hyperemic areas on the conjunctival surface of the upper lid, in which are one or two yellowish pinpoint-sized areas. The symptoms, course, treatment and histopathology are given, and the following summary is made:

"A form of keratoconjunctivitis with adenitis is described which in its protean manifestations resembles many known diseases of the conjunctiva and cornea with which it has been confused but which bacteriologically is shown to be the same disease.

"Pannus in a case of conjunctivitis in man is regarded by most authorities as the strongest evidence that the disease is trachoma. In guinea pigs and rabbits a form of conjunctivitis with pannus occurs naturally; it has also been experimentally reproduced. This pannus differs from the pannus of hypersensitiveness in being unable to produce curtain pannus. The disappearance of the bacteria by small tarsectomies, whereas clinically the granules persist, and the production of greater amounts of fibrous tissue raise an interesting point in trachoma, that probably many cases are spontaneously cleared of bacteria although clinically the signs persist.

"It will be seen that the corneal affections are different from the epidemic superficial keratitis which has been described previously. The monocular phase formed the subject matter of another paper."

W. S. REESE.

THE RESULTS OF CORNEAL TRANSPLANTATION. J. W. TUDOR THOMAS, Brit. M. J. **1:** 114 (Jan. 16) 1937.

The author begins by stating that successful results in corneal transplantation can now be obtained, but conclusions as to the satisfactoriness of the procedure will depend on the publication of detailed reports of all the cases in which this treatment is used.

The cases should be classified according to a definite scheme, and a uniform classification is suggested. This scheme consists in classifying the eyes into three groups: group 1, suitable eyes in favorable patients; group 2, suitable eyes in unfavorable patients and group 3, unfavorable eyes. The results in each group should be given separately. Success depends on the degree of transparency of the graft and the absence of an opaque membrane immediately posterior to it. The results should be analyzed on the basis of the transparency of the graft after a definite period rather than on that of visual acuity.

A summary is given of the results of Elschnig, Filatov, Castroviejo, and Tudor Thomas, and the following conclusions are reached: In order to insure the union of the graft the cutting of the graft shelving, Tudor Thomas' original suggestion, is essential. On the whole, it may be stated that the results in favorable cases were 50 per cent and those in unfavorable cases 38 per cent, and from Tudor Thomas' own figures the chances of obtaining a graft that can be described as transparent are 27 per cent.

The author is convinced that corneal transplantation can now be recorded as a practical procedure, though much is still to be done to make the treatment more effective and to improve the operative technic.

ARNOLD KNAPP.

UNUSUAL CASE OF XANTHOMATOSIS CORNEAE. A. SZÁSZ, Arch. f. Augenh. **110:** 373, 1937.

The right eye of a 43 year old woman showed moderately advanced trachomatous pannus. Several injections of trachoma vaccine were

given. Four years later the patient returned with a yellowish mass over the upper third of the cornea. Histologic examination showed simple granulation tissue. Four years later the patient returned, and at that time a yellowish tumor covered the entire cornea. The tumor was removed and on examination was found to consist of granulation tissue with a varying cellular structure. Some foreign body giant cells were present, and there were a great many cells which had a foamlike structure, due to the presence of droplets of fat in the cells. These cells were xanthomatous. Shortly thereafter the globe was enucleated and examined histologically. Xanthomatous cells were at this time noted throughout the cornea. The author concludes that fats were deposited in the affected tissues as a result of disturbed local metabolism and not as a result of fatty degeneration of the tissue. The main collection of xanthomatous cells was at some distance from the infiltrated region.

F. H. ADLER.

Experimental Pathology

CHANGES IN THE CORNEAL NERVES AFTER EXTIRPATION OF THE GASSERIAN GANGLION IN RABBITS. K. REISER, Arch. f. Augenh. **110:** 253, 1937.

The gasserian ganglion was extirpated on one side in a series of rabbits, and after various periods of time the rabbits were killed and both eyes enucleated. The corneas of these eyes were sectioned and examined for degenerative nerve fibers. None of the rabbits lived longer than seventy hours, but Reiser points out that this period is sufficient for all the signs of degeneration in the nerve elements to appear. He found that after thirty hours the first evidences of degeneration are seen in the large nerve bundles of the cornea. There occur nodular swelling of the nerve fibers and clumping together of many nerve fibers into broad bands. Scattered through these bands are round bodies, apparently the result of fibrillolysis. The more peripheral sections of the corneal nerves showed fewer changes. In the preterminal network there were practically no pathologic changes. The nervous terminal reticulum seemed to remain free from any changes and did not degenerate. This, Reiser believes, is further proof of the syncytial nature of the peripheral nerve elements.

F. H. ADLER.

General

HEREDITY OF SJÖGREN'S DISEASE ASSOCIATED WITH KERATOCONJUNCTIVITIS SICCA. R. LISCH, Arch. f. Augenh. **110:** 357, 1937.

Lisch describes the condition generally referred to as Sjögren's disease in three successive generations. Of thirteen persons examined, eleven showed definite signs of this symptom complex, which consists of dryness of the mouth, nose and throat, difficulty in swallowing dry food and, occasionally, hoarseness. Associated with this occasionally there was absence of tears, with dryness of the conjunctiva. Two patients suffered from filamentary keratitis with diminished corneal sensitivity. In a few of the patients studies of the gastric secretions

showed subacidity. No changes were found in the blood of any of the patients to suggest pernicious anemia as claimed by Grósz. Lisch discusses the possibility of this disease being due to some disturbance in the vitamin metabolism and establishes the familial symptom complex.

F. H. ADLER.

General Diseases

OCULAR CHANGES IN SCHÜLLER-CHRISTIAN-HAND'S DISEASE. C. BEHR, Arch. f. Ophth. **136:** 403 (Feb.) 1937.

Behr reports two cases of the type of lipoidosis which in America is best known as Christian's disease. It consists of the development of circumscribed granulomas with heavy lipoid infiltration in various parts of the body, chiefly in the bones of the skull. Secondarily softening and hemorrhages occur in these granulomas. The outstanding clinical symptoms are maplike defects in the bones of the skull, exophthalmos and diabetes insipidus. The author analyzes the ocular changes which develop in the course of the disease. The exophthalmos may be caused by a lipoid granuloma located either in the bony wall of the orbit or in the sheath of the optic nerve (parenchymatous lipoidosis). The latter form is difficult to diagnose. The diabetes insipidus in Christian's disease is produced by granulomas located in or near the hypophysis, the tuber cinereum or the infundibulum. In one of the cases reported by Behr the patient improved greatly while kept on a diet low in fat. In the other case there was only one orbital lesion, which was removed surgically. No local recurrence and no other lesion developed during the subsequent five years.

P. C. KRONFELD.

Lacrimal Apparatus

CRITICAL STUDY OF THE MEDICOCHIRURGICAL TREATMENT OF DACRYOCYSTITIS. R. ARGAÑARAZ, Arch. de oftal. de Buenos Aires **11:** 567 (Oct.) 1936.

Argañaraz gives a historical review of the treatment of dacryocystitis since the time of Hippocrates to the present day. He considers that drainage of the lacrimal sac is the fundamental factor in the cure of chronic dacryocystitis; the better the drainage, the better the result. Dacryocystitis is due not to a nasal obstruction but to the existence of a closed septic cavity. By opening freely the external wall of the sac toward the inner angle of the eye, free communication is established between the conjunctiva and the lacrimal sac, transforming the latter into a conjunctival culdesac and curing the dacryocystitis but not the epiphora.

After a further study of the origin of the epiphora, the relation of the permeability of the lacrimal secretory apparatus to tearing and the cause of dacryocystitis, Argañaraz considers the indications for probing, excision of the lacrimal sac and dacryocystorhinostomy. He describes his procedure of opening the lacrimal sac through the conjunctiva, combined in obstinate cases with puncture of the bony wall with a special trocar, as a substitute for the two latter operations. He considers the endonasal Toti operation the ideal procedure.

C. E. FINLAY.

Lens

INFLUENCE OF PHLORHIZIN AND SODIUM FLUORINE ON THE VITAMIN C CONTENT (CAPACITY OF REDUCING DICHLORPHENOLINDOPHENOL) OF THE LENS. H. K. MÜLLER, Arch. f. Augenh. **110:** 321, 1937.

The following experiments were undertaken to determine whether the vitamin C content of the lens would be influenced by poisoning animals with phlorhizin and fluorine.

In a series of ten normal rabbits the vitamin C content of the lens was found to be 26.2 mg. per hundred grams, that of the aqueous 31.2 mg. and that of the adrenal glands 299 mg.

A second series of rabbits of the same race, age and state of nutrition were given 0.05 mg. of phlorhizin per gram of body weight twice daily subcutaneously, over a period of twenty-one days. Occasionally some of the poison was also administered subconjunctivally. At the end of the treatment the animals were killed, and the vitamin C content of the lens, aqueous and adrenal glands was immediately determined. It was found that the vitamin C content had diminished in the lens to an average of 16.5 mg. per hundred grams, that in the aqueous to 21.2 mg. and that in the adrenal glands to 199.5 mg.

A second series of animals were given fluorine, and similar studies were made. These animals showed a slight increase in the vitamin C content of the aqueous, lens and adrenal glands.

Müller discusses the various chemical possibilities by which the changes in the vitamin C content may occur. He concludes that in his studies this was due either to some disturbance in glycolysis or to some influence on the permeability of the aqueous-blood barrier.

F. H. ADLER.

Methods of Examination

A NEW ADAPTOMETER. J. LIJO PAVÍA, Arch. de oftal. de Buenos Aires **11:** 341 (June) 1936.

The author refers to a prior paper of his, read in May 1930 before the Argentine Ophthalmologic Society, to a paper of J. Nordman and to one of J. Nordman and P. Payeur, on the adaptation for clinical purposes of Drem's ingenious photometer in common use in cinematographic work. He believes that this device is practical for the following reasons: (1) low cost, (2) facility of adaptation to the eye, (3) rapid working, (4) direct reading of the scale and (5) uniformity of illumination.

C. E. FINLAY.

ENTOPTIC METHODS. E. P. FORTIN, Arch. de oftal. de Buenos Aires **11:** 628 (Nov.) 1936.

Fortin describes in detail numerous entoptic findings with modern illuminating apparatus, which permit an accurate study of the affected parts of the eye, furnishing data often superior to those furnished by ophthalmoscopy. The author cannot understand why this method of examination has been so persistently ignored.

C. E. FINLAY.

CLINICAL EXPERIENCES WITH THE NEW ZEISS PROJECTION PERIMETER (AFTER MAGGIORE). H. SERR, Arch. f. Ophth. **136:** 477 (Feb.) 1937.

The main advantages of the projection perimeter over the ordinary perimeter are: uniform brightness of the stimulus, irrespective of its place of appearance on the arc of the perimeter, and the absence of distracting visual or acoustic stimuli. Perimetry can be carried out with the projection perimeter more quickly and with less fatigue than with the ordinary perimeter. The brightness of the stimuli can be varied so that perimetry on the semidark-adapted eye is possible. The projection perimeter is also suitable for the detection of small central and paracentral scotomas.

P. C. KRONFELD.

Neurology

MECHANISM OF HEADACHE. T. FAY, Arch. Neurol. & Psychiat. **37:** 471 (Feb.) 1937.

Through the application of faradic stimulation traction irritation and heat to the structures within the cranial cavity, with the patient under light anesthesia or with the part under local anesthesia, Fay observed that a sensation of pain similar to localized headache could be produced only when the branches of the meningeal artery are directly involved. Referred pain similar to headache could be elicited by stimulation of the superior sinus from a point approximately underlying the hair line of the forehead to the torcular Herophili, the two lateral and the two sigmoid sinuses. Great pain was experienced from the traction or stimulation of the branches of the venous supply, the tentorium, the inferior straight sinuses and the cavernous sinus and, on one occasion, by stimulation of the right carotid plexus.

Fay noted the following with regard to pain referred to the eyes: Stimulation of the arterial tree near the branches of the circle of Willis produced pain referred deep into the eyes and forehead. Stimulation of the superior sinus produced diffuse pain, regional in character, referred to a point deep in the eyes, especially as the point of stimulation approached the torcular Herophili. Stimulation of the tentorial veins and lateral sinuses produced pain referred to the occipital area and deep behind the eyes. Compression of the jugular vein of the neck, with pressure in the lateral sinus and posterior portion of the superior sinus, produced occipital headache referred deep in the eyes, when the vascular engorgement reached a point of actual gross tumescence of the dural sinuses.

The author believes that his observations show that fibers for pain situated on the large vascular branches of the cranial structures leave the skull, face and head via their vascular structures to descend into the upper part of the thorax by various roots and into the spinal cord through the posterior route mechanism between the seventh cervical and the fifth thoracic segment of the cord.

In summary, Fay reduces the pathogenesis of headache to the common denominator of pain due to stretch of the cerebral vascular tree when extremes of hydration or dehydration are such as to alter

grossly the volume relationships of the cranial cavity and bring about physical stretch on the sheath of the large arteries and venous sinuses. He suggests treating by hydration or dehydration, whichever counteracts the cause of the symptom in the particular instance. He gives his criteria for deciding whether a headache is due to hydration or to dehydration.

R. IRVINE.

OCULAR PARALYSIS FOLLOWING MUMPS. T. HARRISON BUTLER AND A. J. WILSON, Brit. M. J. **1:** 752 (April 10) 1937.

This is a report of two cases of neurologic complications of unusual localization after mumps.

Case 1 is that of a boy of 9 years who, three weeks after he had been taken ill with mumps, showed paralysis of the soft palate and of accommodation. There was slight blurring of the edges of the disks. The knee jerk was absent on one side. Examination for diphtheria gave negative results. When seen again a number of years later the eyes were found to be normal. The case is interesting as an example of the picture of postdiphtheritic paralysis of the intrinsic muscles of the eyes caused by the virus of mumps.

Case 2 is that of a boy of 12 years who, a month after an attack of mumps, had difficulty with vision in the left eye and inability to see anything on the right side. The eyegrounds were normal. There were complete paralysis of the left third nerve and paralysis of the soft palate. There was no evidence of diphtheria. A few days later the left external rectus muscle became affected. The superior oblique muscle, however, remained active. A year later there was still paralysis of the third nerve.

The two cases are of interest because of the type of nerves involved and because of absence of recovery in the second case.

ARNOLD KNAPP.

ARACHNOIDITIS AND VASCULAR COMPRESSION OF THE CHIASM IN A TABETIC PATIENT. M. DAVID, E. HARTMANN and E. HÉBERT, Bull. Soc. d'opht. de Paris, December 1936, p. 789.

The authors report the case of a man aged 48 who was known to be tabetic for four or five years. Gradually there was loss of vision in the lower quadrants of the fields, associated with inability to recognize objects below the usual level of the eyes. Vision was reduced to 2/10 for the right eye and 3/10 for the left eye. General antisyphilitic treatment failed to increase the vision or the fields, but apparently there was no increase in the atrophy of the optic nerve. Exposure of the chiasm revealed optochiasmic arachnoiditis. The anterior cerebral artery was fixed by fibrous strands to the membranes at the crossing. The compression was relieved, and the fields increased markedly in size. Visual acuity was enhanced from 2/10 to 3/10 for the right eye and from 3/10 to 5/10 for the left eye. This is the first reported case in which syphilitic meningitis surrounding the chiasm in a tabetic patient has been treated by operative means. A photograph of the operative field and charts of the visual fields are included.

L. L. MAYER.

Operations

A NEW OPERATION FOR CHRONIC GLAUCOMA. O. BARKAN, Am. J. Ophth. **19:** 951 (Nov.) 1936.

Barkan gives a brief history and critical survey of operations for glaucoma. He refers to his investigations of the cause of glaucoma and states that in over half of the cases of noncongestive primary glaucoma obstruction of the outflow of aqueous from the anterior chamber into Schlemm's canal causes the condition. This obstruction is located in the sclerocorneal trabeculum. He states that these conclusions are confirmed by incising the trabeculum and opening Schlemm's canal, thus restoring the normal tension. This is the basis of his operation, which consists of opening Schlemm's canal by means of a knife introduced at the temporal limbus and carried across the anterior chamber until its blade disappears behind the nasal limbus. He now uses a specially constructed contact glass and a head loupe, which enables him to follow the knife while it pierces the trabeculum and opens Schlemm's canal. He reports eleven cases in which this procedure was used and gives his conclusions and the following summary:

"It is suggested that this operation, which restores the physiological function of Schlemm's canal, solves the surgical problem of most cases of chronic primary glaucoma. It is equally successful in certain cases of secondary glaucoma. The results are predictable and appear to be permanent. It involves a new principle in the surgery of glaucoma in that the angle of the anterior chamber and Schlemm's canal are under full view and magnified during the operation. The operation is without danger when the proper technique is used and has proved completely successful in the writer's hands when certain preoperative indications (binocular biomicroscopic diagnosis) have been fulfilled."

W. S. REESE.

KERATOPLASTY FROM CADAVER'S CORNEA. M. I. AVERBACH, Vestnik oftal. **10:** 345, 1937.

Averbach discusses the advantages of using the cadaver's cornea for corneal transplantation. He recommends the use of the cadaver's conjunctiva (Filatov's method) for a conjunctival flap instead of the host's conjunctiva; by this procedure a large defect in the conjunctiva is avoided, and less trauma is produced in the host's eye. The cadaver's conjunctiva should be saved from injury as much as possible.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

THE OPTIC FORAMEN. J. GRAY CLEGG, Brit. J. Ophth. **20:** 667 (Dec.) 1936.

This article has to do largely with the roentgenology of the optic foramen. Clegg reviews the literature and reports a case of his own, that of a man aged 25. Visual acuity was 6/6 and blurred in each eye. There was a large absolute central scotoma. The foramen

measured 3 mm. in the right eye and 4 mm. in the left. The optic disks were extremely pale.

The author concludes: "Careful and accurate radiography should be carried out in all cases of affection of the optic nerves. Positive evidence of variations in the lumen and in the state of the walls of the optic foramina is of considerable help, but negative findings are also of much service in diagnosis and treatment."

W. ZENTMAYER.

OPHTHALMOLOGIC SEQUELAE IN THE RADICAL CURE OF FRONTO-ETHMOIDAL SINUSITIS. P. HALBRON, Bull. Soc. d'opht. de Paris, October 1936, p. 636.

The ocular complications following operation on the frontal sinus are well known. Halbron reviews such complications in forty-eight patients. These are grouped into four classes: (1) difficulty with motility of the globe, (2) lacrimal complications, (3) palpebral complications and (4) corneoconjunctival complications.

In four of Halbron's patients the complications fall in the first group. Diplopia was the leading symptom. He cautions that treatment must be delayed, as restitution may occur even as late as three months following the operation. Graphs of the muscular balance in these four patients are shown. Interference with lacrimation is most likely due to subcutaneous inflammation. Complications affecting the lid are frequent. Even after healing, inflammation may persist at the internal angle of the lid. Infection of the conjunctiva has been reported, but this did not occur in Halbron's cases. Keratitis without ulceration, however, was frequent.

Halbron concludes that complications may be minimized if the rhinologist keeps in mind the following points:

1. The proper incision avoids the lacrimal sac by passing under and to the side of it.

2. The periosteum of the orbit is removed except in the region of the pulley of the superior oblique muscle.

3. The lids must be held in proximity following the operation.

A bibliography is appended.

L. L. MAYER.

DIAGNOSIS OF FRACTURES OF THE SPHENOIDAL WINGS. K. VELHAGEN, Arch. f. Augenh. **110:** 365, 1937.

Velhagen examined a series of skulls at the Anatomical Institute at Halle and classified the sutures formed by the sphenoid bone as zygomatic and frontal. These were arranged in three different types. The difficulties of diagnosing fractures in this region are mentioned.

F. H. ADLER.

ECTOGENOUS INFECTION OF THE EYEBALL WITH BACILLUS PHLEGMONIS EMPHYSEMATOSAE (WELCH-FRÄNKEL). H. RIEGER, Arch. f. Ophth. **137:** 61 (April) 1937.

Rieger reports five cases of septic endophthalmitis following perforating injury. The condition was characterized by a fulminant course and

by general malaise with fever. By culture of material obtained by puncture of the vitreous Bacillus Welchii was demonstrated to be the cause of the endophthalmitis. In such infections the fulminant course often makes evisceration of the globe necessary.

P. C. KRONFELD.

Physiology

AN INVESTIGATION OF THE ANGULAR RELATION OF THE VISUAL (VISIERLINIE) AND OPTIC (CORNEAL) AXES OF THE EYE. G. E. PARK, Am. J. Ophth. **19:** 967 (Nov.) 1936.

This technical article does not lend itself to abstracting. The following conclusions are stated:

"1. The described method of securing coincidence of the optic axis of the telescope with the visual axis is accurate to within a maximum error of one-fourth degree.

"2. The amount of error in identifying the visual axis by means of the red spot is of the same order as given above.

"3. The angular relation of the optic and visual axes is a variable which accounts for different readings over a series of observations or consecutive readings within a short period.

"4. These variable readings can scarcely be attributed to any corneal change nor to a change in the anatomical position of the fovea.

"5. This leaves but one factor within the eye to account for such variation, the crystalline lens. Possible unequal action of the ciliary muscle, causing prismatic action of the lens by excessive compression at a portion of the lens margin or bodily decentration of the lens from the then existing visual axis, would account for such variation.

"6. The described effect of eserine is entirely upon the ciliary muscle, which seems to indicate this variation to be a result of unequal action of the ciliary muscle.

"7. There appears to be a definite coordination between the ciliary and extrinsic muscles in establishing and maintaining fixation."

W. S. REESE.

CALIBER OF THE RETINAL VESSELS IN HEALTH AND DISEASE. E. LOBECK, Arch. f. Ophth. **136:** 439 (Feb.) 1937.

Lobeck's method for the determination of the caliber of the retinal vessels (*Arch. f. Ophth.* **133:** 152, 1934) is based on the principle of heliometry. Double images of the fundus are produced by cutting the eyepiece of Gullstrand's ophthalmoscope into halves and by letting the half lenses slide against one another in the plane perpendicular to the optical axis. This displacement is done and measured by a micrometer screw. To estimate the width of a part of the fundus one of the images is displaced until the displacement equals the width to be measured. The micrometer readings are transposed into millimeters by substituting 1.5 mm. for the diameter of the disk, which is measured in micrometer scale units in every case. With this method the width of the

first branches of the central artery and vein were measured for a number of normal persons and for patients with various kidney diseases. The latter diseases were classified according to Volhard's classification. Patients with cardiac decompensation were excluded. The results are given in the table.

Caliber of the Retinal Vessels in Normal Persons and in Patients with Kidney Disease

Diagnosis	Width of the Retinal Vessels in Mm.		Relation in Caliber Between Arteries and Veins
	Arteries	Veins	
Normal condition	0.134-0.088	0.176-0.097	1:1 to 1:1.4
Red hypertension	0.127-0.074	0.149-0.09	1:0.9 to 1:1.45
Pale hypertension	0.081-0.053	0.138-0.097	1:1.5 to 1:2
Nephrosclerosis	0.058-0.027	0.131-0.092	1:1.57 to 1:4
Nephrosclerosis with albuminuric retinitis	0.033-0.026	0.095-0.091	
Acute nephritis	0.133-0.071	0.161-0.09	
Chronic nephritis	0.062-0.053	0.112-0.098	1:1.71 to 1:1.86
Chronic nephritis with albuminuric retinitis	0.084-0.057	0.112-0.100	

Lobeck states: "In all cases of albuminuric retinitis we found a disproportion between the caliber of the retinal arteries and veins. This was more pronounced in the cases of nephrosclerosis than in those of chronic nephritis . . . In diabetic retinitis the retinal arteries had a normal caliber." Acetylcholine, choline and aminophylline produced a measurable degree of dilatation of the retinal arteries, whereas amyl nitrite and perhaps also caffeine dilated the retinal veins.

P. C. KRONFELD.

DIRECTED PERMEABILITY OF THE ELASTIC MEMBRANES OF THE EYE. B. NAKAMURA, Y. UCHIDA and O. NAKAMURA, Arch. f. Ophth. **136:** 471 (Feb.) 1937.

In experiments in vitro the diffusion of cevitamic acid through the capsule of the lens was quicker in the direction into the lens than in that out of the lens.

P. C. KRONFELD.

Refraction and Accommodation

SUDDEN SPASM OF ACCOMMODATION (INTOXICATION BY METHYLENE BLUE?). J. PLICQUE, Bull. Soc. d'opht. de Paris, October 1936, p. 617.

A patient aged 37 years consulted Plicque because of sudden blurring of his distance vision. The visual acuity of the right eye declined from 9/10 to 3/50, and that of the left eye from 9/10 to 5/50. The pupillary diameter and reflexes were normal. The ocular tension measured 28 mm. No treatment was instituted for twenty-four hours in order to note any change and for the purpose of diagnosis. After that period vision remained the same; the tension receded slightly. Atropine

was instilled, and refraction carried out with the eyes under the influence of this cycloplegic showed the need for a — 1.75 D. sph. for the right eye and a —3.5 D. sph. for the left, but forty-eight hours later the correction for the right eye was + 0.5 D. and the left eye was emmetropic. The urine and the Wassermann reaction were normal. The question of diabetes and psychophysiologic change was investigated, without positive results. Cases of a similar condition that occurred after an operation for strabismus, reported by Plantenga and by Paton, in a patient with parkinsonism, reported by Guillermin and Chams, and in association with facial spasm, reported by Terrien, Schaeffer and Blum, gave no insight into the cause. The patient had been treated with methylene blue (methylthionine chloride) for gonorrheal urethritis. Plicque questions whether the drug's influence on the neurovegetative system, with predominance of stimulation of the parasympathetic system, may not have caused the condition. A bibliography is given.

L. L. MAYER.

Retina and Optic Nerve

THE LAURENCE-MOON-BIEDL SYNDROME—RECORD OF A CASE. JOSEPH R. MUTCH, Brit. J. Ophth. **21:** 225 (May) 1937.

The patient's mother, one of a family of twelve, is living and healthy; two males are affected with atrophy of the retina and optic nerve. The patient is the third of five children, four being males and one a female. There was no parental consanguinity. The remaining two brothers and the sister are alive and normal.

The patient is aged 35. His vision became dim at the age of 12 years but later improved, only to fail again at the age of 30. Night blindness is not present. Vision is 6/60 in each eye. The fields are contracted and show a defect in the left upper temporal area. There is an indefinite central scotoma for red and green. He is not color blind. Each optic disk is pale and has sharp edges. There are a slight mottling at both maculae and a gray spot near the left macula but no pigmentary changes at the periphery. The other symptoms are: moderate obesity, hypogenitalism and hypospadias, mental retardation, polydactyly and syndactyly. The article is illustrated.

W. ZENTMAYER.

SYNDROME OF DIRECT COMPRESSION OF THE INTRACRANIAL PORTION OF THE OPTIC NERVE. PIERRE DESVIGNES, Ann. d'ocul. **174:** 289 (May) 1937.

As indicated by the title, this article deals with cases in which a tumor, either fibrous or vascular, presses on the part of the optic nerve just in front of the chiasma. The optic nerve is fixed here by its continuity with the chiasma and by the optic canal and the arteries of this region, the ophthalmic artery outside and below and the anterior cerebral artery and the anterior communicating artery above. In such a position the optic nerve cannot escape from pressure, as it might do in the orbit, where it is surrounded by soft tissue. It therefore follows, if a tumor begins in this neighborhood, that the nerve will soon be compressed, and ocular signs will first draw attention to the condition.

The signs of compression of the chiasma are well known, but those of compression of the optic nerve have not been as much studied. In some cases study has established the diagnosis of a cerebral tumor localized near the optic nerve, and in such cases if operation has not been too long delayed patients have escaped rapid blindness and death accompanied with great suffering.

The author gives first a historical review of the subject, followed by a discussion of the first clinical signs and of (1) vision, (2) the visual fields, (3) the appearance of the fundus and (4) other ocular symptoms. There is a bibliography.

S. H. McKee.

A Case of Detachment of the Retina with a Hole in the Macula: Treatment by Diathermy. L. Mamoli, Ann. d'ocul. **174:** 309 (May) 1937.

This article consists of a short report of the case of B. J., 31 years old, of delicate health, who was found to have the retina detached completely in its inferior part. There was bilateral myopia of 13 D., with bilateral macular chorioretinitis. There was present a macular hole across which could be seen the displaced choroid. Because of the much reduced vision, the age of the patient and the presence of the hole, operation was decided on, but the various known technics did not give satisfactory results.

The patient was treated by diathermy coagulation of the macular region, obtained by the use of an electric needle introduced into the interior of the globe and pushed by ophthalmoscopic control to the retinal region.

S. H. McKee.

The Problem of Papillary Edema of Hypertensive Patients. A. Couadau, Bull. Soc. d'opht. de Paris, July 1936, p. 579.

Edema of the disk is the primordial and primitive sign of the papilloretinitis of hypertensive patients. Associated with hemorrhages and exudates of the retina, it completes a typical ensemble which is easily recognized and has a grave prognosis. Couadau has especially studied isolated edema of the papilla. According to its intensity it assumes two forms—simple edema and edema due to stasis. Simple edema is frequently observed, seventeen cases having been under the author's observation in a few years. Edema of the disk due to stasis necessitates differentiating between that due to papillitis of hypertensive patients and that due to a cerebral tumor associated with intracranial hypertension. Two cases are reported in which signs of nephritis were not present in the early course but appeared later. In cases of true tumor the edema of the disk is progressive. No signs of nephritis are ever present. The retinal arterial tension is raised in papillitis of hypertensive patients but fluctuates in stasis.

Summarizing, Couadau feels that differentiation depends on the findings of general examination and the observations of biochemical study and on the progress or course of the condition. A bibliography is appended.

L. L. Mayer.

ANGIOID STREAKS OF THE RETINA AND PSEUDOXANTHOMA ELASTICUM. A. V. DENTI, Ann. di ottal. e clin. ocul. **65:** 93 (Feb.) 1937.

The literature on angioid streaks of the retina associated with pseudoxanthoma elasticum (the syndrome of Groenblad and Strandberg) is reviewed. Denti's patient, a woman of 37, showed a typical form of each condition. Vision was reduced to 1/2. Aside from the typical streaks about the disks, the fundi showed hemorrhages and a number of fine white spots, especially in the central region. Examination of other members of the family gave negative results. During six months numerous fresh hemorrhages were observed in the internal layers of the retina, but none were so deep as the streaks. The author interprets the streaks as abnormal vessels of congenital origin in the deeper retinal layers, perhaps in connection with the choroidal veins. He believes that the degeneration of elastic tissue which is known to cause the cutaneous lesions also causes degeneration in the walls of the retinal vessels and in the lamina vitrea of the choroid, such degeneration being responsible for the hemorrhages and white deposits.

S. R. GIFFORD.

SIGNIFICANCE OF DYNAMOMETRIC STUDIES FOR THE EXPLANATION OF THE MECHANISM OF CHOKED DISK. J. SOBAŃSKI, Arch. f. Ophth. **137:** 84 (April) 1937.

In 1934 Sobański published in Polish a monograph on the mechanism of choked disk (reviewed in *Zentralbl. f. d. ges. Ophth.* **31:** 540, 1934). The theory elaborated therein was based chiefly on dynamometric studies, but was also based on experiments on animals in which pressure was exerted on the orbital portions of the optic nerve by means of a clamp. The dynamometric studies demonstrated that the development of a choked disk is conditioned on a disproportion between the pressure in the central vein and that in the central artery. Normally this relation is 1 : 3. If the intracranial pressure rises, the retinal venous pressure always rises also. If the arterial retinal pressure rises at the same rate no choking develops, despite the increased intracranial pressure. If the arterial pressure does not rise sufficiently so that the ratio between the venous pressure and the arterial pressure becomes 1 : 1.5, papilledema develops. In this article Sobański restates his views and objects to theories based only on pathologic observations.

P. C. KRONFELD.

INFLUENCE OF RED RAYS ON THE FUNCTION OF THE RETINA. H. M. DEKKING, Arch. f. Ophth. **137:** 153 (April) 1937.

The sensitivity for all visible rays of the dark-adapted foveal region of the human retina is lowered by simultaneous incidence of visible rays of a wavelength over 6,200 angstrom units. This phenomenon is most pronounced for minimal intensities of light.

P. C. KRONFELD.

FURTHER OBSERVATIONS ON RETROBULBAR NEURITIS IN THE CHINESE. CHEN P'AN and W. P. LING, Chinese M. J. **50:** 1373 (Oct.) 1936.

The authors report the study of seventy patients with retrobulbar neuritis admitted to the wards from a total number of one hundred

and eighty patients with this condition who were seen. In all the cases the pupils were dilated for examination of the fundi. The findings were normal in all cases except five, in which the patients showed retinal hemorrhages without other changes.

The visual fields were examined carefully; there were no abnormal findings. A central scotoma was practically the only sign of retrobulbar neuritis present in all the cases and was usually a relative one for both white and colors; it was confined to the fixation point and was not larger than 10 degrees all around.

The authors conclude that the etiologic factor of retrobulbar neuritis in the Chinese still remains a mystery. They believe that the term "retrobulbar neuritis" applied to this condition may be misleading and think that the phrase "retrobulbar changes of the optic nerve" is preferable. The disease runs a tedious course; the prognosis is usually good, no case of blindness having been met with as yet. There is no special treatment for the condition, and the general condition of the patient is, as a rule, good.

S. H. McKee.

Pseudo-Atrophy of the Optic Nerve in Young Infants. K. T. A. Halbertsma, Nederl. tijdschr. v. geneesk. **81:** 1230, 1937.

Halbertsma reports the clinical history of a girl 8 weeks old who was brought to him by the parents with the complaint that the eyes had a peculiar staring expression which had made them fear that the child might be blind. The ophthalmologic examination revealed an abnormal behavior of the pupils and a peculiar coloration of the optic papillae. On the basis of another examination made three weeks later, the disorder was diagnosed as pseudo-atrophy of the optic papilla probably resulting from retarded myelinization of the pupillomotor fibers of the optic nerve. Several successive examinations disclosed gradual improvement in that the pupillary reactions became more normal and the peculiar coloration of the papillae began to disappear. The author points out that the condition in this case corresponds to that described as pseudo-atrophy of the optic nerve in the new-born by Beauvieux in 1926. He also cites the signs which, according to Beauvieux, differentiate pseudo-atrophy of the optic nerve from true congenital atrophy of the optic nerve.

J. A. M. A. (W. Zentmayer.)

Trachoma

Subconjunctival Autohemotherapy in Trachoma. E. S. Martinez, Rev. internat. du trachome **14:** 19, 1937.

In two cases of trachoma in which from 2 to 4 cc. of the patient's own blood was injected under the fornix the ocular irritation rapidly subsided and the pannus was favorably affected.

J. E. Lebensohn.

Trachomatous Keratitis. S. Spyrâtos, Rev. internat. du trachome **14:** 23, 1937.

The statistics of Athens show a definite recrudescence of trachoma and trachomatous keratitis in October, probably owing to intercurrent

catarrhal infections. As the patients grow older the proportion of corneal complications steadily increases.

J. E. LEBENSOHN.

CONSTITUTION IN RELATION TO TRACHOMA. F. MASSOUD, Rev. internat. du trachome **14:** 42, 1937.

Very young children acquire trachoma more readily than adults. Lymphatic children suffering from adenoids seem most susceptible. Special sanatoriums for persons with trachoma have been established in Italy, in which the value of sunshine, fresh air and proper food is emphasized.

J. E. LEBENSOHN.

THE CONTAGIOUSNESS OF TRACHOMA CLINICALLY CONSIDERED. J. SÉDAN, Rev. internat. du trachome **14:** 65 (April) 1937.

Trachoma is especially contagious in infants, but contagion is possible in persons of all ages, though it occurs seldom in children of school age and only rarely in adults. Important predisposing factors are neglect in personal hygiene, intercurrent conjunctivitis, disease of the lacrimal apparatus and ocular traumatism. Patients with trachoma in boarding schools, barracks and hospital or hotel service should be closely supervised or excluded. The incidence of trachoma is greatest where the general standard of living is lowest.

J. E. LEBENSOHN.

TRACHOMA AND INFECTIONS OF THE LACRIMAL PASSAGES. A. ALIQUÒ-MAZZEI, Ann. di ottal. e clin. ocul. **65:** 38 (Jan.) 1937.

Aliquò-Mazzei examined three hundred and eighty patients with trachoma with especial reference to involvement of the lacrimal passages. In only 12 per cent of the cases was there moderate interference with the passage of the fluid into the nose, and in only 5 per cent of cases was there complete obstruction. These were cases of old trachoma and extensive scarring of the lids, with obliteration of the canaliculi or tear points. Dacryocystitis was present in only 4.21 per cent of the cases. In these cases (sixteen) the lacrimal sac was removed. Thirteen patients showed the inflammatory changes commonly seen in dacryocystitis, with the presence of follicles. In three cases true follicles were present in the walls of the sac. There was nothing characteristic about the history. The author concludes that the dacryocystitis did not have any causal relationship with trachoma.

S. R. GIFFORD.

Tumors

CONJUNCTIVOPALPEBRAL TUMOR (DERMOLIPOMA) OF EXCEPTIONAL DEVELOPMENT IN A NEW-BORN INFANT. M. A. DOLLFUS, Bull. Soc. d'opht. de Paris, October 1936, p. 598.

In an otherwise normal new-born infant a pedunculated tumor was noted outside the outer canthus. It was red at birth but on twisting of the pedicle assumed a violet color. It was the size of a large olive, regular, palpable and mobile on the cheek. The conjunctival stalk appeared to have its origin in the cul-de-sac, but a few strands con-

tinued to within a millimeter of the corneal limbus. Pathologic examination confirmed the clinical diagnosis of dermolipoma, as the many and various blood vessels, the sebaceous glands and other dermal elements would testify. A drawing is included. The exceptional size and location are the cause for the report.

L. L. MAYER.

TUMORS OF THE ORBIT: ORBITOFRONTO-ETHMOIDAL OSTEOMA. Q. DI MARZIO, Riv. oto-neuro-oftal. **13**: 393 (Sept.-Oct.) 1936.

Di Marzio describes four cases of orbital osteoma located chiefly in the frontal sinuses. In the first case he made a linear incision from the root of the nose along the superior margin of the orbit as far laterally as the tail of the left eyebrow, continuing the incision from the head of the left eyebrow across the glabella to the head of the right eyebrow. The periosteum was separated; the bone was attacked with a hammer and chisel, and excavation was made down to the internal angle of the left frontal sinus, to the root of the nose and along the arch of the orbit, where the tumor was strongly attached. Since the tumor was very large and invaded the frontal sinuses, it was necessary to separate it from its superior wall; the dura was almost completely exposed, pulsating but not perforated.

The tumor had invaded the left as well as the right frontal sinus, extending toward the ethmoid sinuses. The cavities of the sinuses were accurately curetted, and drained by way of the left nostril and the original orbital wound. The deep layers were sutured with catgut, the superficial layers with silk. The tumor, which was found to be an osteoma, measured 5.5 by 5.2 by 4.5 cm. and weighed 68 Gm.

In the second case, since the mass was confined chiefly in the inner angle of the left eye, Di Marzio made a curvilinear incision from the external third of the superior orbital margin to the region of the lacrimal sac, detaching the periosteum down to the ethmoid sinus, where the neoplastic mass was located, and then down to the lacrimal fossa, and separating the lacrimal sac. With a hammer and chisel he then removed the floor of the frontal sinus and the wall of the ethmoid sinus so as to cut out the tumor, which was closely adherent to these structures. He sutured the periosteum and skin, and applied drainage through the nose. This tumor was smaller than that in the first case, measuring 3 cm. in every diameter.

In the third case the tumor was located on the floor of the right frontal sinus and extended down to the ethmoid sinus. The technic of the operation was the same as in the second case.

The fourth case was interesting because six years previously the region over the left eye had become swollen and the patient had fever. At that time he was operated on for orbital abscess, but the exophthalmos did not regress. Four years later the eye again became swollen: the swelling subsided following hot applications.

Roentgen examination at this time revealed a dense opacity in the region of the left orbital margin, which extended to the right orbital margin, invading most of the left orbit and part of the frontal bone through the frontal sinus and reaching up to the anterior cranial fossa. The patient was operated on, with the part under local anesthesia an

incision was made along the left eyebrow and down to the bone, and the external wall of the frontal sinus was chiseled out. A large lobate mass was seen adhering to the inner wall of the sinus. The rest of the cavity was filled with pus and granulation. The tumor was removed piecemeal with a bone forceps, a very thin long sheet being left, through which the meninges could be seen pulsating. The granulations were removed by curettage, and the sinus cavity was carefully cleaned and drained through the nasofrontal canal and through the exterior wound. This tumor was large, measuring 6 by 4 by 4 cm.

Di Marzio stresses the value of complete removal of the tumor and curettage of the mucosa of the sinuses in order to prevent the inflammation from spreading from the sinuses to the cranial cavity.

He discusses the pathologic features of the neoplasm and concludes, agreeing with Lagrange and Borst, that osteoma of the orbit may have several origins, which must be distinguished in each case

G. BONACCOLTO.

Vision

VISUAL TASKS IN SIGHT-SAVING CLASSES. M. LUCKIESH and F. K. Moss, Am. J. Ophth. **19:** 992 (Nov.) 1936.

This article is based on the determination of actual visual efficiency. Luckiesh and Moss state that the criterion of visual acuity is faulty, whereas measurements of visibility are fundamentally sound. Size of type as a factor in seeing was appraised by measurements of visibility made with the Luckiesh-Moss ophthalmic sensitometer. The authors state that by their technic it is possible to classify pupils in sight-saving classes into a limited number of groups and provide each group with type of the proper size.

W. S. REESE.

Vitreous

THE IMPORTANCE OF DISEASES OF THE CHOROID FOR DEVELOPMENT OF DETACHMENT OF THE VITREOUS. H. RIEGER, Arch. f. Ophth. **136:** 119 (Nov.) 1936.

To the long series of articles dealing with detachment of the vitreous published by pupils of Lindner, Reiger adds a clinical study of 356 persons suffering from diseases of the fundus in which detachment of the vitreous is known to be common. In addition to a thorough ophthalmoscopic examination of the fundus, the vitreous was examined ophthalmoscopically and with the slit lamp, with and without a contact glass. The data thus obtained indicate that detachment of the vitreous is common in, and probably caused by, diseases of the choroid. A large number of interesting and new slit lamp pictures of detached vitreous are described.

P. C. KRONFELD.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OPHTHALMOLOGY

April 15, 1937

CHARLES R. HEED, M.D., *Chairman*

ALEXANDER G. FEWELL, M.D., *Clerk*

ANESTHESIA INDUCED WITH TRIBROMETHANOL IN OCULAR SURGERY.

DR. EDWARD W. BEACH.

Tribromethanol when used to induce anesthesia in surgical intervention on the eye reduces the psychic shock because it allows administration in the patient's room and produces a quiet sleep. It relaxes the extra-ocular muscles and reduces the intra-ocular tension. The slight fall in the blood pressure which it causes minimizes bleeding during operation and gives a paler conjunctiva than other anesthetics. When employed as a basal anesthetic it requires much less of the supplementary agent than other anesthetics. The recovery of consciousness is prolonged, and this period of amnesia is welcomed by patients with ocular conditions. Because of slow recovery there is need for a longer period of nursing to prevent unconscious movements. There are little or no postoperative nausea and vomiting.

DISCUSSION

DR. CHARLES W. LE FEVER: I have used much larger doses of tribromethanol than those just mentioned by Dr. Beach. I started using smaller doses, averaging from 75 to 80 mg. per kilogram of body weight, but soon found that it was necessary to give the patient ether to complete the anesthesia. This small dose is sometimes sufficient for senile patients. In patients under 40 years of age, of the muscular type, I give 95 mg. per kilogram, and even then it is often necessary to give supplementary ether.

I have never seen any especially unfavorable effects from tribromethanol. In older persons I believe it is the rule that the blood pressure falls rapidly during the first twenty minutes, while the patient is becoming narcotized. At the end of that time, or perhaps within thirty minutes from the time the tribromethanol was given, the drop in the blood pressure stops. Thereafter the blood pressure starts to rise, continuing to do so gradually from this time until the completion of the operation or until it reaches normal. I have seen it fall as low as 56 systolic and 20 diastolic, at which point it responded promptly to 5 minims of epinephrine hydrochloride given by hypodermic injection.

DR. LUTHER C. PETER: I have been using tribromethanol for a number of years and find it one of the best forms of anesthetic for

surgical intervention on the eye. In fact, it has few limitations, as Dr. Beach has pointed out. It is not satisfactory for a child under 10 years of age, because of inability of the patient to cooperate. The upper age limits have not been fixed thus far. I agree with Dr. Beach, however, that elderly persons and those who are obese should receive the smaller dose of 80 mg. rather than the upper limit of 90 mg.

In the matter of advantages and disadvantages, Dr. Beach has placed lowering of the blood pressure and lowering of the intra-ocular pressure as disadvantages. On the contrary, I regard these effects as advantages in cases of cataract and glaucoma. Among the disadvantages, nausea is the one which must receive careful consideration. In my experience in the Graduate Hospital of the University of Pennsylvania, there have been few cases of nausea, and in most of these nausea was due to a necessarily long period of anesthesia, as in operations for retinal detachment, when supplementary anesthesia must be used. Ether is not a satisfactory supplementary anesthetic. Divinyl ether and chloroform are the best agents of this kind.

The great advantage of using tribromethanol in operations on elderly persons, especially those who have cataract, is the fact that it eliminates worry. The anesthetic is administered in the patient's room. A second point of great advantage, especially in the more difficult operations, is the quietness of the patient on the operating table. In fact, tribromethanol has made surgical intervention a real joy, even in the most difficult cases. I refer to cases of complicated cataract following uveitis, in which there are marked posterior synechiae. One can make the section without difficulty, release the adhesions with a spatula and deliver the lens in its capsule without any difficulty. A bridle suture is introduced under the superior rectus muscle to control the eye.

The other type of case in which tribromethanol is advantageous is that of the patient who has had a sclerocorneal trephining performed and who also requires extraction of cataract. As is known, the anterior chamber, as a rule, is shallow. But with a narrow cataract blade the section can be made and a conjunctival flap fashioned; the wound can be enlarged by straight scissors; synechiae, if present, can be released, and the lens can be extracted in its capsule. In fact, when the patient is perfectly relaxed and the eye is quiet one can do almost any type of difficult surgical intervention.

CHOROIDEREMIA: REPORT OF A CASE. DR. WALTER LILLIE.

Arthur J. Bedell in his article on choroideremia (ARCH. OPHTH. **17**: 444 [March] 1937) stated:

"The subject of this essay is a peculiar condition characterized by night blindness, sometimes evident in early life, but often not conspicuous until the third or fourth decade. It seems to be familial, affecting the males. . . . Choroideremia is an entity with a pathognomonic fundus, and as it develops during the life of the patient it must not be considered as a congenital absence of choroid but as a dissolution of that membrane. . . . A large portion of the choroid is absent; the macula is intact, and the vessels of the choroid are small and straight or

entirely absent. There is no ectasia of the sclera. . . . The macular region is the last to register the destructive process."

Report of Case.—A man aged 70 years was referred to me by Dr. F. W. Uhler, of Easton, Pa., because of diplopia for the past three months. The patient had always been in good health. For twenty years he had worn bifocal lenses with satisfaction. During the past three years he had been able to carry on as road supervisor without difficulty, although he was conscious of scintillating scotoma in the form of "heat waves," and when the bright sunlight was reflected from buildings "red vision" resulted. His glasses were satisfactory, although ocular fatigue ensued after half an hour of reading. One year previously, during a period of three months he lost 20 pounds (9.1 Kg.), but he had been well since October 1936. My examination, on Feb. 23, 1937, revealed vision to be 6/10 in the right eye and 6/12 in the left eye, with his glasses. The correction for the right eye was a + 0.12 D. sph. combined with a + 1.00 D. cyl., ax. 92, and that for the left eye was a + 0.25 D. sph. combined with a + 0.75 cyl., ax. 90, with an additional + 2.50 D. for near work on each lens. The pupils were equal and the reflexes normal. The ocular movements revealed slight weakness of the left internal rectus muscle and loss of convergence. The ophthalmoscopic examination revealed a few small opacities in the vitreous in each eye. The optic disks were normal in color, and the margins were distinct, but there was loss of the choroid in the peripapillary area. The retinal arterioles were slightly attenuated. The choroid was absent in the lower portion of the fundus in each eye and in an area extending into the superior nasal portion in the right eye. The border was distinct, and the retinal arteries extended uninterrupted over this area. Clumps of pigment were fairly numerous but well scattered. A few choroidal vessels still remained, which disappeared abruptly into the normal choroidal margin. The sclera appeared normal. The macular region in each eye was intact. Tests of the visual fields revealed definite bilateral superior altitudinal anopsia. The color vision was normal as tested with the Ishihara charts. The general examination revealed nothing of importance, and the patient's health was excellent for this age group. There had apparently been no similar familial visual disturbance in the past two generations. This case is of interest, as it not only furnishes an additional instance in the small group of cases of choroideremia already published but apparently is the case in which the visual disturbance had the oldest onset, with no night blindness, "red vision" from reflected sunlight being a disturbing feature. The diplopia was probably a coincidental condition of the central vessels of the retina.

PARTIAL BILATERAL COLOBOMA OF THE OPTIC NERVE. DR. FRANCIS HEED ADLER.

Complete coloboma of the optic nerve seldom offers any difficulties in diagnosis. The porus opticus is at least two or three times the diameter of the normal disk, and the cup is often 3 or 4 D. in depth. Associated with anomaly of the disk there is frequently a coloboma of the choroid. In cases of partial coloboma of the optic nerve, however, there may be

errors in diagnosis. A case of partial coloboma of each optic nerve was reported, in which the condition had been diagnosed as primary optic atrophy and the patient had been referred to Dr. Francis Grant as possibly having a pituitary tumor.

COLOBOMA OF THE CHOROID AND PARTIAL COLOBOMA OF THE DISK. DR. ALEXANDER G. FEWELL and DR. EDWARD S. GIFFORD JR.

J. K., aged 8, presented in the right eye a congenital crescent on the nasal side of the disk and a hole in the disk on the nasal side. Below the disk was a large coloboma of the choroid, oval and located at 180 degrees, separated by a narrow strip of normal fundus from another choroidal coloboma below which it extended into the periphery as far as could be seen. In the left eye there was a small, round coloboma of the choroid, extending down and out from the disk.

M. K., aged 16, a sister of this patient, showed a hole in the disk on the temporal side in the left eye.

The fundi of each parent were normal, the mother being hyperopic and the father myopic. Of 6 living children, record of only 4 could be obtained. The 2 just cited as presenting anomalies were hyperopic; 2 others, who had normal fundi, were myopic.

REVIEW OF GLAUCOMA AT THE WILLS HOSPITAL, PHILADELPHIA. DR. LOUIS LEHRFELD and DR. JACOB REBER.

This article will appear in a later issue of the ARCHIVES.

A STATISTICAL INVESTIGATION OF SYPHILITIC ATROPHY OF THE OPTIC NERVE. DR. LOUIS LEHRFELD and DR. ELMER R. GROSS.

A ten year survey of syphilitic atrophy of the optic nerve at the Wills Hospital included study of 552 patients with primary atrophy of the optic nerve and 48 with secondary atrophy of the optic nerve. Of these 600 cases, 91 were studied and observed sufficiently for conclusive evidence to be obtained. The remaining cases were tabulated for statistical purposes. The purpose of this survey was to determine what happened to these patients with syphilitic involvement of the optic nerve from a therapeutic, prognostic and social-economic standpoint.

The patients were divided into three main groups. Group I consisted of those patients who had received no treatment for the atrophy of the optic nerve. The patients in this group were blind or nearly blind on admission to the clinic. Group II was composed of those patients who had received antisyphilitic treatment. Group III was made up of those patients who had received a special form of treatment, including fever and subdural therapy.

Seventy-four and nine-tenths per cent of the patients in group I were blind in less than three years, and all were blind at the end of a five year period. Twenty-three and eight-tenths per cent of the patients in group II were blind in less than three years, and all were blind at the end of eight years. Twenty-eight per cent of the patients in group III were blind in less than three years, and all were blind at

the end of eight years. This refers to the observed cases from the onset of the symptoms to blindness. It appears that primary atrophy of the optic nerve is a progressive process, despite treatment. The present day therapeutic armamentarium does not offer a great deal with which to combat this grave complication of syphilis.

A general statement cannot be made that treatment is contraindicated in primary atrophy of the optic nerve; although not 1 patient was considered improved in our series, the condition in 8 remained stationary with routine treatment and that in 11 remained stationary with subdural treatment. It is, however, likely that these patients may be blind at the end of a ten year period.

The result of the subdural treatment depends primarily on the irritant effect of the substances introduced intraspinally. A modification of the Swift-Ellis treatment that we employ in the clinic at the Wills Hospital seemed to produce more meningeal irritation, which manifested itself in signs and symptoms of aseptic meningitis. The patients who have had these reactions seem to hold their own better than those who have not.

The onset of atrophy of the optic nerve in most instances is so insidious that the person does not realize the seriousness of the situation. When the patient complains of blurring of vision, the atrophy of the optic nerve is usually already pronounced. The early concomitant signs and symptoms of the associated neurosyphilis are so mild that they frequently escape the physician's attention.

The present day treatment of syphilitic primary atrophy of the optic nerve is unsatisfactory. As in all forms of syphilis, prophylaxis is the best form of treatment. This can be accomplished by education of the public and the physician. Measures to prevent atrophy of the optic nerve should include, first, early diagnosis and adequate treatment of early syphilis and, second, early diagnosis and adequate treatment of neurosyphilis. Pupillary abnormalities are often the first objective signs of neurosyphilis, and any patient in whom these are observed should be subjected to a thorough examination, including a test of the spinal fluid. The diagnosis and treatment of atrophy of the optic nerve require close cooperation between the syphilologist and the ophthalmologist. Plotting of the visual fields, examination of the fundus and tests of visual acuity are of prime importance in diagnosis and in determining the severity of the atrophy of the optic nerve.

DISCUSSION

DR. LOUIS LEHRFELD: The reports presented by Dr. Gross and Dr. Reber demonstrate that all research in ophthalmology is not confined to the laboratory. At the Wills Hospital there are many volumes of records which are waiting for investigators such as Dr. Gross and Dr. Reber to delve into their stored-up knowledge and to abstract from them information which may be just as important to ophthalmology as researches made in the laboratory.

Dr. Reber's report states that in 22 per cent of the patients with glaucoma who reported at the Wills Hospital in the last ten years the condition was secondary. Generally, one dismisses secondary glaucoma as playing no part in the consideration of the subject of glaucoma.

Our investigations led us to believe that the causes responsible for secondary glaucoma give a strong hint as to the pathologic physiologic condition incident to primary glaucoma.

Dr. Gross' survey points out the ineffectiveness of the present methods of treating atrophy of the optic nerve that is the result of syphilitic infection. His report indicates that the ophthalmologist must look in other directions for newer methods in collaboration with the syphilographer.

Both these investigations should give encouragement to the younger ophthalmologists to carry out research based on clinical records coupled with examination of the patients whom these records concern.

EFFECT OF DISTANCE USED IN TESTING ON THE APPARENT SIZE OF A SCOTOMA. HAZEL WENTWORTH, PH.D.

The effect of the distance used in testing on the apparent size of a scotoma, as indicated by changes in the size of 100 normal blindspots for form and color when outlined at different distances from the eye, was described.

The exact area of the blindspot in 100 different eyes was obtained for 1 degree Hering white, Heidelberg blue, red and green test objects at distances from the eye of 16.6, 33 and 100 cm., respectively, and exactly measured in an effort to determine whether the distance from the eye at which a scotoma is mapped has any effect on its apparent size, that is, on the visual angle subtended, and, if so, to what extent, and whether there is any differential color effect.

The work was done on the Holloway-Cowan screen under carefully controlled conditions as to the illumination, the brightness of the background, the method of mapping, the size of the test object, the amount of practice and other factors.

With the blindspot obtained at 33 cm. taken as the standard size for comparison, it was found that the size of the average blindspot increased relatively at the distance of 16.5 cm. and decreased relatively at the distance of 100 cm. The total decrease in size for the average blindspot at a distance between 16.6 and 100 cm. from the eye was 3.5, 8, 22 and 27 per cent for form, blue, red and green, respectively, as compared to a mean error in size for the same number of repeated tests at the same distance (33 cm.) of 2.5, 2.8, 3.3 and 3.4 per cent, respectively.

From tables showing the distribution it was found that for red and green 8 or 9 of every 10 subjects showed an average decrease in size of nearly one third that taken as the standard when the distance was increased from 16.6 to 100 cm. from the eye; for blue, 2 of 3 subjects showed an average decrease of one fourth the standard area, and for form slightly over one half showed an equal amount of change. Only about four fifths as many patients showed a decrease in size when the distance was increased from 16.6 to 33 cm. from the eye as when it was changed from 33 to 100 cm. The changes in apparent size with changes in distance were considered, on theoretical grounds, to be chiefly the result of changes in accommodation. They were shown to

be valid from the statistical standpoint. The causal factor of the differential color effect could not be definitely determined.

The significance for practical perimetry was pointed out, i. e., the fact that not only does the area of a scotoma obtained at different distances from the eye represent different retinal areas when a constant visual angle is assumed but also, since the change is differential for form and color and between the colors, the relative areas of partial and complete loss of sensitivity vary at the different distances. The factor of distance was shown to be of particular importance in the use of a red test object, since red is the color showing the greatest change with distance and a test with this object is also the most reliable sensitive test of lesions in the optic nerve fibers and tract which are not observable on objective examination. The importance of the differential effect is shown in that, when attempting to determine the extent to which the nerve fibers have become involved in a retinochoroidal lesion, as indicated by the relation of the limits of the scotoma for red and for blue, respectively, the area of the scotoma for red will be judged relatively smaller as compared to that for blue at the greater distance. A plea was therefore made that for comparable results a standard distance be adopted.

This paper, presented in tribute to the late Dr. T. B. Holloway, who suggested the problem and whose support made it possible to carry out the work, will be published in full at a later date.

DISCUSSION

Dr. Francis Heed Adler: Just what causes the change that Dr. Wentworth has outlined no one can say. Whether accommodation is an important factor could perhaps be determined by examining a number of patients under the influence of a cycloplegic.

Dr. Sidney L. Olsho: Before scientific conclusions can be drawn I should like to ask if it would not be necessary to insure that the lighting be so controlled that the test objects receive the same amount of illumination at the two unequal ranges, that of the campimeter and that of the tangent screen.

Dr. Hazel Wentworth: The illumination of the screen was kept constant at 7 foot-candles at all distances. The Holloway-Cowan screen was designed to make this possible.

Dr. Walter I. Lillie: If the size of the blindspot decreases at increased distances it might be of interest to know whether the peripheral field varies at similar distances.

Dr. Hazel Wentworth: No similar studies have been made of the peripheral field. I have found it difficult at times, however, to decide whether or not a certain increase in the amount of contraction of a field as the distance increased was definitely tubular.

Dr. Alfred Cowan: I should like to know whether changes in the pupil were taken into consideration, as these might possibly be the factor.

Dr. Hazel Wentworth: Changes in the pupil were considered as a part of the general accommodative effect.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

London, England, June 11, 1937

MR. W. H. MCMULLEN, O.B.E., F.R.C.S., *President*

THE EPITHELIAL GROWTHS OF THE CONJUNCTIVA AND CORNEA. MR. E. F. KING.

There seems to be no essential difference in the pathologic nature of the epithelial growths found on the conjunctiva and on the cornea. The tumors may be either benign or malignant. The usual site for malignant neoplasms is the limbus. The more innocent tumors may assume malignant properties.

Of the innocent tumors, the first is papilloma, which is seen mostly in the fornices and on the caruncle. Each papilla consists of a delicate stroma containing thin-walled vessels, covered by stratified epithelium. This type of tumor shows a tendency to local recurrence after it has been removed, thus resembling similar tumors in the urinary tract, and it may become malignant. Another form of innocent tumor is the epithelial plaque, which occurs on the conjunctiva or the cornea as a well defined area of epithelial hyperplasia, superficially keratinized. Parsons considers the plaques congenital and dermoid in nature. Lister and Hancock regard them as analogous to callosities on the surface of the skin, representing hyperplasia of the horny layers.

Of the malignant growths, rodent ulcer never involves the eye primarily, but only secondarily by extension. The common site for epithelioma is the limbus, but a number of cases have been reported in which it arose wholly on the cornea. Epithelioma on the conjunctiva, at a distance from the limbus, seems to be the result of malignant change in a papilloma. This variety of tumor is most frequently seen in the interpalpebral space, especially on the temporal side, that most exposed to trauma. In several cases this tumor has followed penetrating wounds. The growth is sometimes pigmented by the normal pigment cells at the limbus and at first resembles a limbic dermoid, but at a later stage it ulcerates and is liable to bleed on slight provocation. Bowman's membrane offers considerable resistance to invasion by tumors, but yields later, and then infiltration occurs along the lymph spaces around the corneal lamella. The sclera forms a dense barrier and becomes eroded rather than infiltrated. On reaching Tenon's capsule the growth extends rapidly, and in some cases it completely surrounds the globe. In a high proportion of cases in which the growth is advanced there is intra-ocular extension along the anterior perforating vessels and Schlemm's canal, or by way of the vorticose vessels in cases in which Tenon's capsule in involved. There seem to have been only a few authentic reports of generalized metastasis.

Microscopically, the growth consists of stratified epithelium and stroma in papillomatous formation. The following features are characteristic of malignancy: (*a*) There is variation in the size and chromatin content of the epithelial cells, in some of which the nucleus is fragmented and degenerate, active mitosis being seen in others. (*b*) Penetration of epithelial cells into the underlying tissues is unusual, a

more usual appearance being actual erosion and loss of substance of the cornea and sclera, without infiltration. Some degree of dipping of the epithelium at the normal limbus is normal. (*c*) Characteristic nests of cells are present, which are seen in the fibrous stroma of the growth rather than in the underlying structures. (*d*) There is inflammatory cellular infiltration in and around the mass, even when there is no secondary infection due to ulceration.

With regard to the treatment of carcinoma, when the cornea is extensively involved or the growth has penetrated the globe, excision or exenteration is clearly indicated. If the growth is an early small limbic tumor it is generally agreed that conservatism is permissible, though many urge excision in all cases if the other eye is sound. As to conservative lines of treatment, local excision, cauterization and roentgen therapy (alone or in combination) have resulted in variable success. Radium therapy seems to offer an efficient and simple treatment which is completely successful in most cases and appears to be particularly indicated for this condition, of which an early diagnosis is the rule. Several authorities have published highly satisfactory results of treatment of epibulbar growths by radium. The method of treatment has been, in general, the application of unscreened beta rays to the surface of the growth for short periods, and this is repeated at intervals, if necessary. If the growth is considerable, it is wise to remove first the greater part of it surgically.

Radiation cataract has been reported by many observers after radium has been used in or near the eye, but there seems to be a great variation in the dose needed to produce this cataract and in the time of its onset.

DISCUSSION

MR. T. HARRISON BUTLER: I remember seeing a tumor with a pedicle, which on biopsy was reported as hemangioma, a variety of nevus. Certain sprays of growth were seen going into the cornea, and I thought that feature suggested malignancy. The patient was sent for treatment to the Radium Institute, and after two applications of radium the condition was cured. In another case the view of the late Treacher Collins and other authorities was that the growth was epithelioma. It was pigmented but was only slightly raised above the general surface, though it implicated the cornea to a distance of several millimeters. It is about twelve years since I first saw the patient. She had several applications of radium at the Radium Institute, and during the twelve years there has not been a suggestion of recurrence.

Mr. James asked me to see a man who had been subjected to roentgen treatment for a long time in order to cure an extensive nevoid condition on his face and in whom a typical radiation cataract had developed. So far as I can see, there is no difference between the cataract caused by the roentgen rays and that caused by radium, if those agents are injudiciously used over a considerable time. I think the risk of causing cataract by using the doses needed in these cases is infinitesimal.

MR. A. D. GRIFFITH: The fear in connection with these cases is the risk of causing radiation cataract. Has Mr. King used much heavier doses of radium in these cases for a shorter period of time, say, 30 millicuries for half an hour, and is this not much less likely to cause

cataract? In one case in which the latter ensued the first thing I noticed was a change in the refraction, and six months later cataract began.

Mr. E. F. King: Technically, the method I have been using is as simple as that carried out at the Radium Institute; it can be repeated when necessary. As to the employment of a heavier dose for a shorter time, I was guided in the matter of procedure by the advice of Mr. Stanford Cade; if I observe a similar case in the future I shall put the point before him and ask his views. His feeling about the lens was that with the doses of radium used in these cases there was little risk of producing cataract.

GERMAN OPHTHALMOLOGICAL SOCIETY

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Docent M. Bücklers, M.D., Tübingen, *Reporter*

Translation by Percy Fridenberg, M.D., New York

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W. Meisner, M.D., Cologne, *Chairman*

DISCUSSION OF REPORT IV

(Continued from page 489)

Dr. O. Marchesani, Munich: As to the interpretation of the term blindness, I think it corresponds largely to that of incapacity (*Invalidität*) of visual origin. It is clear that we wish to prevent the progeny not only of blind subjects but of incapable subjects as well. If we have recourse to the conception of incapability, this offers the advantage of a concept which is familiar to all ophthalmologists. The statement "One must anticipate that with the greatest probability in the following generations there will be hereditarily diseased subjects" is not to be understood in the sense that these probabilities must necessarily be determined in each individual case. This would lead to perfunctory or intentionally motivated decisions. On the contrary, the commentary attached to the general provisions of the sterilization law state definitely that this great probability is presented in all cases of hereditary disease which is known to be transmitted according to either the dominant or the recessive mode. The recognition of a disease of this nature is sufficient. In our opinion, zonular cataract frequently originates in factors of the environment (*Umwelteinflüsse*). Slitlamp studies frequently show definitely at just what period of intra-uterine life or at what age the damage was done. There is to my mind no contradiction between the old view that this disease is due to rickets and the prevalent opinion that tetany is the etiologic factor of prime importance. For it is well known that in

the healing stage of rickets there is a disturbance of metabolism which results in a general spasmophilia. So rachitis is not so much the cause of zonular cataract as it is an indicator. The question arises whether we are not to envisage the cases of hereditary zonular cataract and those of zonular cataract which has developed on a basis of spasmophilia from a uniform standpoint. According to von Pfaundler—and subsequent research in cases of identical twins has supported this view—the tendency (*Anlage*) to rickets is itself hereditary (rachitic diathesis).

It is probable that this *Anlage* may be inherited in varying degrees, quantitatively. In the cases of manifestly hereditary zonular cataract the tendency alone would be decisive, whereas in the cases of other types of zonular cataract an additional factor, that of upset metabolism, is requisite for the manifestation of the disease.

DR. WISSMANN, Wiesbaden: Does the practical ophthalmologist merely have to report patients for sterilization, or is it part of his function in the evaluation of the condition to make a detailed scientific report and, in a way, to place at the disposal of the court (*Erbgericht*) his own opinion as an expert to serve as a basis for the decision?

DR. ENGELKING, Heidelberg: It would be a mistake to minimize the difficulties which may present themselves in the practical carrying out of the sterilization statutes. A warning is in place that the diagnosis is not to be considered the only, or even the predominant, leading datum. For instance, not every patient with retinitis pigmentosa should be certified for sterilization without further consideration. There are, without doubt, families in which this condition is hereditary and hereditable (*vererbbar*) and in which the members through several generations have maintained fairly good vision for life. Just as little does every patient with total color blindness deserve to be sterilized, as there are patients with this condition, as I can report from my own experience, who have perfectly good sight and are practically undamaged. Some congenital hemeralopes are quite capable of gainful occupation. These, too, in my opinion, need not be sterilized. Another difficulty is presented by the fact that certain diseases appear now as hereditary and again as nonhereditary. This applies, for example, to microphthalmos, which may occur in connection with microcephaly by external traumatism, or by damage to the fetus, such as that caused by the roentgen rays. The same is true of coloboma in association with defective sight. In these and analogous disorders it is my opinion that sterilization should be called for only when positive proof of the hereditary character of the condition is forthcoming, reserve being maintained in cases in which the condition occurs sporadically (*isoliert*).

DR. W. CLAUSEN, Halle: The enormous value of the sterilization statutes can be fully grasped only from the standpoint of general medicine and racial hygiene, not merely from that of ophthalmology alone. Ninety per cent of those to be sterilized are subjects with a congenital mental defect, and in such cases a decision surely ought not to be difficult. Of the remaining 10 per cent, those who are to be sterilized on account of hereditary ocular disease represent only a very small fraction. I have sharply defined the concept of practical blindness as far as it applies and is possible for the application of the law. It corresponds,

one might say, with a visual disability so extreme that incapability (*Invalidität*) is caused by it. Each case must be considered separately and evaluated from every angle. It goes without saying that heredity must be definitely established and that the report of examination and the history must be definite and complete and the diagnosis correct. It is highly probable that more intensive research in the immediate future will tend to show that glioma is hereditary in many more cases than has been assumed heretofore. In the past the question of possible inheritance was not looked into thoroughly, as glioma was not considered hereditary until quite recently. In regard to total color blindness, I have called attention to the fact that it is generally associated with nystagmus and visual defect of the highest degree. Only such types of total color blindness and not those in which vision was approximately normal were considered.

INDIVIDUAL PAPERS

I. Parenchymatous Keratitis. Dr. J. Blaickner, Salzburg.

On the basis of the observation that patients with interstitial keratitis present, in greater percental frequency than patients of the same age with other conditions, anomalies in the development of the thyroid gland which are verified by inspection and palpation, the basal metabolic rates for 34 patients with this condition, who were observed in 1933-1936, were determined according to the method of Krogh and evaluated in relation to the body weight and the body size in accordance with the standards in Benedikt's tables. In 3 of these cases the determination of the basal metabolic rate could not be carried out on account of non-cooperation and fear on the part of the patient, but in all the other cases reliable curves for breathing were obtained. The values found varied between + 10 and — 10 per cent in 15 cases, but it is to be noted that by far the greater number of these values lay below the zero point. However, on account of the mathematical limit of error of about 10 per cent, which is the general allowance, this entire group is reckoned as normal.

The figures were between — 10 and — 20 per cent in 6 cases, between — 20 and — 30 per cent in 2 cases and between — 30 and — 40 per cent in 5 cases, whereas there were only 4 cases in which the values were between + 10 and + 36 per cent. Naturally, an attempt was made to check up these figures with those for controls. Determinations in 10 control cases gave the following figures: In 4 cases the values were between — 10 and + 10 per cent; in the other 6 the values were between + 10 and + 32 per cent. Of the patients with parenchymatous keratitis, 11 were between 4 and 10 years of age, 13 were between 11 and 20 and 9 were between 21 and 30, and 1 patient, a woman, was 36 years old. Eighteen of the patients were males and 16 females. In his material the reader was unable to find any relation between the low basal metabolic rate and the sex or the age of the subject. On the other hand, the few patients with a high metabolic rate were all under 15 years of age. The results of these investigations, then, with all necessary critical reserve, show a definite preponderance of negative values, i. e., minus figures; but in this connection it must be borne in mind that even possibly

very slight diminution in the basal metabolic rate, which in a given case cannot be accurately determined on account of the comparatively wide limits of error inherent in the method, may be decisive if one considers the highly complicated cell metabolism in the cornea—a nonvascular tissue—particularly when one is dealing with a pathologically altered or destroyed cell life. On the basis of the results of these clinical tests, treatment with thyroxin was added to, and combined with, the invariably and thoroughly carried out antisyphilitic therapy. One or two ampules were given weekly ("i. m." [intramuscularly?—P. H. F.]), depending on the degree of lowering of the basal metabolic rate. No shortening of the course of the disease could be noted, but there was a definite change in the clinical course.

If the patient had been treated from the very beginning with thyroxin in addition to the antisyphilitic therapy, it was striking that in all the condition frequently and regularly ran the same course, which remained unchanged for a long time. This condition was characterized by Igersheimer as the type of parenchymatous keratitis with posterior or deep deposits, and separate, scattered foci of opacity in the deepest layers of the cornea, immediately in front of Descemet's membrane. These posterior deposits often persisted obstinately for a long time, but the final result was, nevertheless, a favorable one, as the middle layers remained unscathed throughout a wide extent. The reader is of the opinion that more importance should be attached to disturbances in the metabolism of the thyroid gland as an etiologic factor in parenchymatous keratitis, although that does not indicate, by any means, that he denies the importance of anaphylaxis. The main factor must, of course, be seen in the active syphilis effective somewhere in the organism, possibly on the posterior surface of the cornea.

II. Keratoconjunctivitis Sicca—Partial Symptom of a Major Syndrome. Dr. Henrik Sjögren, Jönköping, Sweden.

As to the symptomatology of this disorder, the reader refers to a monograph by him, published in 1933 in the *Acta Ophthalmologica*. General examination revealed the following details: 1. The sedimentation (*Senkungs*) reaction showed increased figures in all the cases. 2. The blood picture was, with few exceptions, characterized by lymphocytosis; in isolated cases there was a low degree of eosinophilia and in a larger number a medium degree of anemia. 3. The body temperature was subfebrile for a longer or a shorter period in all the patients, 11 in number, who were examined. These reactions indicate that one is dealing with a chronic infection. The pathologic changes in the conjunctiva consisted in a high degree of atrophy of the epithelium. The lacrimal glands as well as the glands of the nose, mouth, larynx and pharynx, were markedly sclerosed, and these glandular changes came on earlier than the lesions of the mucous membrances. Keratoconjunctivitis sicca is, accordingly, a partial symptom of a larger symptom complex which depends on infection and which when fully developed presents, in addition, xerostomia, rhinopharyngolaryngitis sicca and chronic polyarthritis. The various symptoms described as combined in this syndrome were formerly, as far as they were known, considered as separate, individual disorders, and there were only speculation and theory as to their

etiology. It is possible, of course, that there are various forms of atrophic rhinitis, differing in etiology. But these investigations prove that in one form, at least, they may occur as indications of a larger symptom complex, that in such cases they depend on an endogenous infection which attacks the glands and that the diminished secretion caused by the disease of the glands brings about the pathologic changes in the mucous membranes.

DISCUSSION

DR. W. LÖHLEIN, Berlin: In Berlin we have made it a principle, long ago, to apply the bengal rose (fluorescein) test in every case of obstinate bacterial inflammation of the conjunctiva, especially in patients at the menopause, and have found keratoconjunctivitis sicca frequently in combination with one or another of the symptoms which Dr. Sjögren has summed up in his monograph. I should like to discuss in detail a typical case in which the subject showed the entire symptom complex in its fullest and completest expression, as it indicates how severely the patient—a woman, as is almost always the case in this condition—was plagued.

The patient, aged 55, the wife of a colleague, had been treated in every way, but without effect, for an annoying and obstinate conjunctival catarrh with stringy secretion and varying opacities and plaques (*Flächenbildungen*) in the cornea. She suffered extremely from unbearable itching, and from blinking, as she could never keep her eyes open for any length of time, and had very annoying and painful sensations after reading or using the eyes for other work at short range for even a short time. The test with fluorescein was positive, giving the characteristic picture, and on closer investigation we learned that the patient had had parotitis in childhood and that this had recurred several times. Besides, she had had dryness of the buccal mucous membrane for years and could not eat without drinking at the same time. The teeth had become brittle, and sensitive swellings had developed about the finger joints. Without going into the details of the general physical examination, which gave essentially negative results, it may be mentioned that there was great relief from contact glasses, which were worn and well tolerated for three or four hours and in this space of time completely relieved the annoying symptoms, as in the moist chamber the epithelial lesions disappeared, and with them insufferable itching. It was very instructive to note the results of the fluorescein test before and after the wearing of the contact glasses. In the first case the lower two thirds of the conjunctiva bulbi and of the cornea were closely sown with reddish points in the epithelium. After the contact lens was removed the test was absolutely negative for the entire area which had been covered and protected by the glass. For the exposed portions beyond this area the test was still positive and showed the old superficial lesions. For severe conditions of this sort, accordingly, one can advise the careful selection of a contact lens.

DR. MARCHESANI, Munich: Dr. Weve has informed me that conjunctivitis sicca is favorably influenced by instillations of fibrolysin solution. The results of the use of this medicament in one case of this condition in our own experience confirms his therapeutic observation.

Third Scientific Session

Tuesday, July 7, 1936, 8: 30 a. m.

DR. LINDBERG, Helsinki, Finland, *Chairman*

III. THE AGING OF THE LENS. HANS KARL MÜLLER, M.D., Basel, Switzerland.

The lens is, above all organs of the body, that which has the greatest independence, as is shown particularly by the organ-specific action of lens albumin parenterally administered. Senile cataract, i. e., the death of the lens due to age, can, accordingly, be nothing else than the death of any given living organism as the result of age. In order to construct a basis for a rational medicamentous therapy for cataract it is essential to determine first the processes which are important for the life of the lens and whether they are within the lens itself or in its more or less immediate neighborhood. Then one must study the development and changes in these processes with age. The central point of therapy for cataract is the recognition of the dependence of the functional development of the lens on age conditions. On this principle, closer studies were made of glycolysis in the lens. These showed that aqueous solutions of lens substance have the property of changing added hexosediphosphoric acid into triosephosphoric acid. When the former chemical was added, the lenticular property of forming esters of organic substances with phosphoric acid was proved. Furthermore, if glycerin phosphate is added to aqueous solution of lens substance, phosphorus is split off. In lenses of old cattle both the aforementioned reactions were present only in a slight degree when the experiment was conducted under similar conditions. However, the property of splitting organic phosphoric acid compounds seems to be maintained at its former level. These and other senile changes explain why the lenses of aging animals contain decidedly less carbohydrate phosphates than those of younger cattle. With Lohmann's hydrolysis process it can be shown that especially the difficultly hydrolyzable fraction of the carbohydrates diminishes with age. This is probably to be attributed to a gradual disappearance of the phosphoglyceric acid. This finding also explains the diminution in the vitamin C content of the aging lens. It was shown that rabbits which had been intoxicated for fourteen days with large doses of phlorhizin had only a small amount of vitamin C (15.9 mg. per hundred cubic centimeters) in their lenses, while the lenses of control animals fed with sodium fluoride for ten days had a very high content (37.6 mg.). Normal animals of the same age showed a vitamin C content of 25.9 mg. in the lens. These reactions to phlorhizin and sodium fluoride, respectively, make it appear probable that phosphoglyceric acid is a precursor of vitamin C. These chemical investigations throw light on the functional changes which take place in the lens in the course of aging and if carried further may lead the way to a future medicamentous therapy of opacities of the lens and cataract.

DISCUSSION

DR. JESS, Leipzig: It is fortunate that we have in our midst research scientists who are willing to devote themselves to the difficult investiga-

tions in the borderland of physiologic chemistry and, of late, of the vitamins and hormones. Twenty years ago when I began to busy myself with physiologic chemistry a prominent clinician informed me that these questions were of slight concern for ophthalmologists. There is no doubt that this old view was a mistaken one, and that if we are to get any nearer to the solution of the problem of medicamentous treatment of lenticular opacities we must, unconditionally, make use of the ways and means placed at our disposal by physiologic chemistry and all the exact methods of natural science. I need only refer to the papers announced for the next few days on the formation of cataract, epithelial bodies, and an irradiated ergosterol preparation which may well be of special significance in the question of the medicamentous effect on opacities of the lens.

Dr. Reinhard Braun, Rostock: Dr. Müller has called attention to the organ specificity of lens albumin as cited, in his time, by Uhlenhuth, and has laid stress on this factor as one of the most important bases of modern research on the chemistry of the lens. I cannot let this statement pass unchallenged and uncontradicted. Numerous experiments have proved to my entire satisfaction that, at least in regard to the anaphylactic components, it is quite permissible to entertain doubts as to the organ specificity of lens albumin. Lens matter alone is quite capable of producing toxic effects which in their further development have an extraordinary similarity to anaphylactic reactions.

Dr. Hans Karl Müller, Basel, Switzerland: In answer to Dr. Braun's remarks, the peculiar individuality of the lens does not depend on the organ-specific action of parenterally administered lens albumin alone. As a matter of fact, even if the organ specificity of lens albumin is not complete, one has still to consider the anatomic and physiologic independence of the lens. This is definitely expressed in the fact that the lens has no blood vessels and is not in direct connection in any way with the central nervous system. Furthermore, although the nutrient fluid of the lens, i. e., the aqueous, contains barely perceptible traces of albumin, the lens is able to organize its chemical composition, probably by metabolic processes, in such a way that it finally has an albumin content of 35 per cent. From this and similar examples one may recognize beyond a doubt that the lens has an independent faculty of biologic development far beyond that of any other organ in the body.

IV. The Problem of Electric Cataract. Dr. W. Comberg, Rostock.

The research in this study was carried out in collaboration with Dr. K. Quest. In cases of injury by lightning and/or currents of high intensity, as well as in experimental studies of electric cataract, it has been shown that very often a severe iridocyclitis complicates the clinical picture. Up to the present time the question to be settled is whether the cataract is essentially due to the passage of the high voltage current through the lens and its capsule, or whether, on the other hand, it is caused secondarily by the severe inflammation of the uveal tract. The reader attempted to solve this question by making a change in the experimental conditions from those in the earlier usual course of research. This consisted in applying the electrical discharge in the region of the

orbital margin of rabbits or placing the electrode directly on the orbital margin and passing the current through the eye, essentially, in the equatorial direction. The reader placed one electrode on the middle of the cornea, using as a second electrode a clip, which was inserted behind the globe; the latter was kept dislocated and proptosed during the experiment. With this arrangement the anterior pole of the lens gets the strongest effect of the current, while the middle portions of the iris and the ciliary body get comparatively little. In the majority of the cases, after subjection of the lens to the current, an opacity developed at the anterior pole, directly underneath the capsule or in the anterior cortex and in the neighboring deeper layers. Even with this new arrangement of the experiment irritation due to iridocyclitis was frequently noted. The opacities of the lens tended to remain stationary, or they increased but slightly in the course of the following few months. The rapid development of the opacities of the lens in the subcapsular region, which had been exposed to the most direct and intense current, indicates strongly that one is dealing without much doubt with a direct action of the electric current alone. Another fact which seems to prove that there was no essential cooperation of an iridocyclitis in these experiments is the clinical observation that in those cases in which uveitis was noted, later progress in the opacities of the lens was as slight as in the cases in which iritic irritation had been minimal.

V. A Fourth Type of Phakomatosis. Dr. J. van der Hoeve, Leyden, Netherlands.

The term phakomatosis was proposed by the reader as a collective concept, derived from φακός, taken to indicate a birth mark or congenital nevus, for syndromes presenting symptoms of a congenital *Anlage*, in which heredity is a factor, for example, the syndromes of Bourneville (tuberous sclerosis of the convolutions of the brain), von Recklinghausen (multiple neurofibromatosis) and von Hipple and Lindau, all of which consist in spots or flecks (phakoi), tumor-like malformations (phakomas) and actual neoplasms (phakoblastomas) complicated by congenital anomalies. In accordance with Albrecht's classification of the origin and genesis of tumors, phakomas may be put into the following categories: (1) choristomas, which are malformations arising from cells scattered in a kind of diaspora far from their usual site, and (2) hamartomas, which develop from autochthonous cells usually found, normally, in the same location, from unused cells which have remained in situ, from embryonal interstitial connective tissue, and so on. The term hamartosis is rejected, because this includes only a part of the symptoms. A picture of a phakoma of the retina in a case of phakomatosis (von Recklinghausen) was sent to the reader by Pascheff, and in the microscopic sections of an eye of one of the patients with Bourneville's phakomatosis which the reader reported in 1921 not less than four retinal phakomas were found. These probably developed by way of internal metastasis via the vitreous, as the reader showed in 1923. A diagram was presented in which the phakomatoses are divided into phakoi, phakomas, and phakoblastomas. The first group comprises congenital birth marks of the following types: (1) phakoi with nevus cells, (2) homotopic phakoi and (3) heterotopic phakoi. The phako-

blastomas, which are true neoplasms, may be benign or malignant. The phakomas, finally, which are tumor-like formations of anomalous histologic and biologic provenance, include: (1) choristomas, which originate from disseminated germinal cells, e. g., hypernephroma of the kidney; (2) hamartomas, which originate from varieties of cells which are normally found in situ, for instance, adenoma sebaceum, (3) phakomas which originate from remnants of unused or quasi-superfluous cells, e. g., neurogliocytoma, and (4) phakomas which originate from embryonal interstitial connective tissue, for instance, glioma as a starting point for syringomyelia. Further, in a phakoma of the nerve head and in a retinal phakoma in a case of Bourneville's phakomatosis, calcified spots were found. The reader placed in the category of the fourth type of phakomatosis Sturge-Weber's disease, which has the following symptom complex: nevus vinosus, buphthalmos, vascular anomalies and angiomatosis of the choroid, calcifications in the brain, epilepsy, idiocy, paralyses, acromegaly, adiposity, atrophy of the brain, angiomatous proliferations in the pia mater and other conditions. He reported a new case, that of a girl aged 3½ years with a very extensive port wine mark of the face, hemiplegia, mental retardation, glioma of the retina, calcifications in the brain and crossed paralysis (on the right, cerebral, and on the left, cerebellar). Deposits of calcium were found only in the brain, not in the cerebellum; accordingly, deposits of calcium and atrophy are, as Krabbe also claimed, independent of one another. The calcium is not bound to the blood vessels, as Bergstrand and others believed. In this brain, which was studied by Prof. B. Brouwer, the calcifications were quite independent of the vessels. The reader pointed out that the plates of calcification in this brain and those in the intra-ocular phakomas in cases of Bourneville's phakomatosis are deceptively similar, even to the point of being indistinguishable, while similar calcifications are also found in the tumors of the ependyma of the ventricles which are generally present in Bourneville's disease. It would appear that this variety of calcification is a symptom quite independent of the phakomatoses. Gliomas are seen in all four varieties of phakomatosis; viz., in the brain in Bourneville-Recklinghausen and/or von Hippel-Lindau disease, and in the retina in the syndrome of Sturge-Weber. There are transitional manifestations from one variety of phakomatosis to another, buphthalmos being seen in Recklinghausen and Sturge-Weber's disease, and retinal phakomas in all forms. There are suggestions of, and analogies (*Anklänge*) with, Hand-Schüller-Christian's disease and with Recklinghausen's bone disease, in the latter case probably owing to the presence of a phakoma in the parathyroid glands. None of the phakomas is limited to any one definite germinal layer. The reader laid stress on the great practical importance of a study of the phakomatoses for increasing knowledge of the structure of the brain and of the pathogenesis of tumors. He also hailed the advance of the knowledge of this topic in the last twenty years, the increased efficiency of therapeutics and the importance of diagnosing the so-called formes frustes (abortive or atypical forms).

DISCUSSION

DR. E. SEIDEL, Jena: Although Dr. van der Hoeve took heredity into consideration in his interesting remarks, I should like to inquire

whether he has carried out any intensive and extensive studies of a genetic nature in this comparatively large series of what are, after all, relatively rare clinical conditions. The subject is of particular interest because in the cases of retinal angiomatosis which I have observed inheritance and/or familial occurrence was noted repeatedly. Thus, in my original communication on the subject to this society (*Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch.*, 1912, p. 335), in which I was the first to call attention to the cerebral symptoms or complications of angiomatosis retinae, I recorded the fact that the brother of my patient with angiomatosis retinae had died, like the latter, as a result of cerebellar cyst. Later I reported (*Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch.*, 1932, p. 535) that both children of the first patient with angiomatosis of the retina who was seen by von Hippel, a daughter and a son, were affected by angiomatosis retinae in each eye. The daughter died as a result of cerebral complications, so I was able to make a microscopic study of the eyes, each of which showed multiple retinal tumors, which I demonstrated at the time at Leipzig. Recently I observed a third case of this disease with familial occurrence, at Jena. The father had an angiomatosis of the retina associated with cerebral symptoms. The family history showed that some years previously a daughter had been operated on elsewhere for a cerebellar cyst and had died subsequently.

DR. FLEISCHER, Erlangen: In addition to the specific and characteristic tumor-like changes in the retina, for which, however, one must search carefully, as they may be very fine and minute in some cases, one notes other lesions. Especially in the peripheral portions of the fundus, one finds small, round, white atrophic foci which at first give the impression of being old atrophic postinflammatory changes but which are undoubtedly to be considered as atrophic foci on account of the characteristic changes in the retina. They are certainly of definite importance for the diagnosis of tuberous sclerosis.

DR. J. VAN DER HOEVE, Leyden, Netherlands: The little white foci in the retina in cases of Bourneville's disease may be of various provenance: They may be small areas of choroiditis, possibly degenerated phakomas, or they may be phakomas in the incipient stage, which may later on develop and grow. It is probable that there are many patients with phakomatosis in institutions, hospitals for persons with mental diseases, and so on, but to us the ambulant patients are more important, those who are going about and who show few striking symptoms, if any, those with the formes frustes, such as the patient already mentioned, the woman belonging to a family in which phakoma was present, who showed only one symptom, viz., a small retinal tumor, while, on the other hand, one of her children was seriously ill. The factor of heredity plays a rôle in all phakomas, and the problems it presents are very difficult. The mother of this girl had asked me whether her daughter's condition was hereditary and whether the latter could marry, as she did not want the daughter to go through the miseries that she herself had endured. After consulting with the neurologists I felt that, although there was a very small tumor-like formation in the retina, I did not have the right to hinder marriage. Up to the present time we know practically nothing about the hereditary character of this disease. If several children of this woman show this disease it will

be, to some degree, my fault. And this consideration again makes me realize how serious the problems are which the questions of heredity now put up to German ophthalmologists. When I came here it was my firm conviction that it is not permissible to sterilize on account of ocular disease alone, and that conviction has not been shaken by all that I have learned yesterday and today from the discussions as well as from the papers. In my opinion, we should exercise the greatest possible caution in this matter, for various reasons. For me, the supreme law for the physician is *primam non nocere,* and one does not know how seriously one injures the patients physically and, above all, psychically; one cannot foresee how great the influence will be of such a procedure, which is of itself not serious but which may well have a serious effect on an organism already damaged by the disease. My second reason for objection is that at present we know far too little about this entire momentous subject to be entitled to go ahead ruthlessly. We must surely recognize and admit that genetics as a science is still in its infancy, however highly we value much of the work that has been done in this field. Very little, on the whole, has been definitely established. We do not even know how sex is transmitted as a heritage. We say that the female has two x-chromosomes and that the male has only one and in addition one y-chromosome. Incidentally, the only possibility of inheriting a sex-linked variation is that the variation be linked to the y-chromosome, and then, surely, only the male sex would be liable to it. From the hypothesis of x-chromosomes and y-chromosomes it follows that just as many males as females would have to be born. However, we know that figures still show that more males are born, so that the theory has not been definitely proved. We may, possibly, get proof from a study of variations obtained from the sex chromosome. For that reason I agree heartily with Franceschetti that it is unquestionably essential to investigate thoroughly the family tree of all color-blind subjects. Yesterday there was mention of a manifest heterozygosity in Leber's disease, which we know to be a sex-linked recessive disorder. This is, in my opinion, a contradiction in terms. In the most extensive family tree of dominant heredity thus far studied, viz., that of Jean Nougaret in hemeralopia, the figures do not correspond at all with the theory, and an attempt is made to explain this discrepancy by the assumption that perhaps the spermatozoa which were afflicted with this variation heritage were less fruitful than others. The path of inheritance is entirely unknown in the case of glioma as yet. In regard to aniridia, which is a typically dominant inheritance, many cases occur as solitary, i. e., nonfamilial. There is much talk of so-called irregular dominance and so on, which probably means merely that the facts and the theory do not fit. We do not know yet, for example, what the percentage of cases of hereditary blindness is. As long as our ignorance is so great, I think we should exercise the greatest reserve as to active procedures and interfere only in cases in which such action is absolutely necessary. And as I am convinced that such interference is rarely, if ever, necessary for patients who have ocular anomalies only, my opinion is now, as it was previously, that, considering the present state of our knowledge, obligation to sterilize on account of ocular defects alone is not permissible.

VI. Diabetic Retinitis. Dr. C. Mylius, Hamburg.

In the course of the last four years 362 of the patients admitted to the hospital for persons with diabetes were also examined ophthalmologically. In 59 of these we found retinal changes which stand in a causal connection with the systemic disease. Those patients were intentionally disregarded in this analysis who had retinal lesions of another sort, such as retinitis proliferans, thrombosis of the central retinal vein or embolism of the central retinal artery, in which perhaps some dependence on the existing diabetes might have been thought of. In all previous papers which have had to do with diabetic retinitis, the factor of high blood pressure has been featured prominently, again and again. In fact, Volhard stated quite definitely—seven years ago, it is true—that he did not know of any cases of diabetic retinitis without increased vascular tension. If we scan the clinical histories of our large group of patients with normal retinas and compare them with those of the group with retinitic changes we must admit on the basis of this parallel that the blood pressure is essentially lower in the first group than in the second. For this several factors are authoritative: 1. In the group with retinal changes the patients were almost without exception elderly subjects—the two youngest in this group were 37 years old. 2. It cannot be explained away that, even if we compare like age categories, the blood pressure in the second group, i. e., that of patients with positive pathologic retinal findings, is, on the average, higher than in the group with normal fundi. Naturally, that alone does not prove anything, for the moment, as to the etiologic significance of high blood pressure in the pathogenesis of diabetic retinitis. We must now call attention to the fact that, in spite of the generally higher blood pressure in the patients with lesions of the fundus, there were no less than 15 in whom the blood pressure was quite normal or even definitely lowered. So it was evident that these, above all, were the patients who called for our especial attention. A most intensive and careful clinical study, with use of all methods of examination, demonstrated that of these 15 patients 8 would have to be excluded, as they were useless for purposes of comparison on account of various intercurrent conditions, such as cardiac decompensation and other severe complications, including carcinoma, tuberculosis, marked emaciation or subsequently increasing values for the blood pressure. We think we can state definitely, however, that in the remaining 7 patients the existing lower levels of the blood pressure are actually to be considered those which we must take into account. Further, it was found that in all these cases we were dealing with type 1 and/or 2 of the categories which Grafe had set up on a basis of Hirschberg's definitions, viz., a condition characterized by fine white stippling and large white plaques, with more or less numerous sharply outlined hemorrhages, particularly at the posterior pole, absence of retinal edema, and normal disks with clearly defined, regular margins. Examination of the vascular system of the retina in these cases showed without exception slight irregularity and variations (*Schwankungen*) in the caliber of, and a somewhat accentuated light streak on, the arteries and, without any essential exceptions, a change in the relation of the diameter of the arteries to that of the veins, of about 2:4. Marked changes in the walls of the vessels were completely missing in these cases, whereas they are very frequently found in group

3 of Grafe's schematic division. These 7 patients were also found to be uniformly free from albuminuria and had, as far as it could be tested, normal renal function. In type 3 of Grafe's category, just referred to, there are always renal complications, and these are made evident not only by the presence of albumin in the urine and marked increase of the blood pressure but regularly, also, in the ophthalmoscopic picture by the appearance of cloudy margins of the disk, retinal edema and marked changes in the walls of the vessels to the point of complete obstruction. We are, accordingly, convinced that we cannot consider these 7 cases in which the blood pressure was low as exceptions which prove the rule. On the contrary, in these instances we are actually dealing with early cases of diabetic retinal disease which must lead us to look at the so often misapplied symptom of increased blood pressure as what it really is, viz., a frequent manifestation running parallel with the retinal disease, one which may be missing in the early stages of that disease and which, accordingly, has nothing to do with the pathogenesis of the retinitis. Further observations tended to corroborate this view. First, in a patient with a perfectly normal blood pressure and normal eyes a bilateral retinitis developed in the course of five years. Second, in 2 cases of retinal lesions which did not belong to the group of the 7 related cases just cited the rise in the blood pressure was observed quite later than the retinal lesions, and certainly did not precede their appearance. As circulatory changes similar to those which we judge to be at the bottom of retinal vascular disease are found in other organs, the increased blood pressure proves to be, as it may be, for the greater part, in our cases, a phenomenon of somatic compensation and regulation. This rejection of increased blood pressure as a systemic, symptomatic factor to be considered in the pathogenesis of retinitis diabetica must not be interpreted and applied incorrectly. For if, on the basis of our observations, we look on the rise in the blood pressure in many of our cases as a compensatory phenomenon, it is logical to conclude that there must be conditions calling for and requiring such compensation, and these are, above all, vascular and circulatory disturbances. The frequency and practical clinical significance and importance of these circulatory complications did not become clear in its fullest extent until after the discovery of insulin. In former times most diabetic patients died of the disease itself, i. e., in coma. Today Joslin's dictum holds true that the diabetic patient dies not of the disease but of the complications, and among these are to be reckoned the circulatory and vascular complications particularly. Even at the present time the change from a diet rich in fat and albumin to one markedly free from these ingredients and rich in carbohydrates has not been carried out generally and consistently. It will have to be worked out gradually. It lays less stress on normalizing the blood sugar than on elimination of ketone bodies, the aim being to prevent the formation of these dangerous substances by maintaining sufficient glycogen reserves in the organism.

(To be continued)

Book Reviews

L'oculistica di Antonio Scarpa e due secoli di storia. By G. Ovio. Two Volumes. Cloth. Pp. 2,296, with 202 figures in the text and 48 plates. Naples: Casa Editrice Libraria V. Idelson, 1935-1936.

Scarpa's name has always been associated with anatomy and surgery. Few know, however, that the "surgeon of Pavia," as Scarpa was called, like his teacher, Morgagni, was much interested in ophthalmology and that in 1801 he published his first book, "The Principal Diseases of the Eye." His clear style, accurate observation, clinical judgment and well balanced application of the principles of pathology known in his day made him a leader at the beginning of the past century.

This masterly work ran into many editions and was translated into German, Dutch, Spanish and English. It became a popular textbook on ophthalmology, in spite of the fact that many books on the subject in various languages were already in existence. About the middle of the past century the book was supplanted by more up-to-date and extensive treatises, and it was almost forgotten.

Professor Ovio, of the University of Rome, has done a service in bringing Scarpa's book on the diseases of the eye to the attention of ophthalmologists. He has taken Scarpa's original work as the main theme and has added to it a history of ophthalmology covering a period ranging from one hundred years before Scarpa's time up to the present.

Professor Ovio has divided his two volumes into two parts. The first part, which is rather short, is dedicated to the general pathologic and therapeutic conceptions of the period during which Scarpa lived; the second part, which is far more voluminous, covers Scarpa's work and is divided into twenty-two chapters.

The chapter dealing with the lacrimal apparatus, although not the longest in the book, is no doubt one of the most interesting. It is a masterpiece of anatomic description and clinical observation. The ingenious use of the so-called "Scarpa's nail" for lacrimal fistulas is described at length. This chapter is rich in the opinions of other ancient writers and of more modern authors and is profusely illustrated, giving the reader a complete comparative study of the subject from its obscure origin up to the present. No doubt even the well qualified plastic surgeon is much impressed to read of Scarpa's criteria on blepharoplasty and other surgical procedures on the lids. Although Scarpa was a skilful operator, he did not use Daviel's operation for extraction of cataract. Ovio comments that Scarpa did not follow this method; he preferred depression of the lens into the vitreous for so-called hard cataract, while he advised laceration of the anterior capsule for soft cataract. He was living in an age in which anesthesia and antisepsis were unknown. He had perhaps seen so many tragic sequels of hazardous extractions that he had become conservative.

His observation on staphyloma, with a consideration of its differentiation from keratoconus, is impressive and is worthy of the pains that Ovio takes in amplifying it with numerous historical and anatomicopathologic annotations. Tumors of the orbit are treated at length.

Scarpa, having been primarily a surgeon and a pupil of Morgagni, was expected to discuss such a topic fully.

In contrast to the topics mentioned, the subject of amaurosis is not very clearly explained. Still one must remember it was about fifty years after Scarpa's book was published that Helmoltz gave ophthalmologists the means of examining the fundus oculi.

Professor Ovio's two volumes are extremely interesting, being enriched with numerous plates representing various ocular conditions, and instruments used at the time of Scarpa, as well as before and after his time. The author has compiled a bibliography of some two thousand references, ancient and modern, which should be an excellent aid to the student of ophthalmology in his critical historical researches.

Ophthalmologists everywhere owe a debt of gratitude to Professor Ovio for these two valuable volumes, which should be in every library of works on ophthalmology.

G. BONACCOLTO.

Ophthalmoscopy, Retinoscopy and Refraction; with a New Chapter on Orthoptics. By W. A. Fisher, M.D., F.A.C.S. Fourth revised edition. Price, $2. Pp. 210, with 240 illustrations, including 24 colored plates. Chicago: H. G. Adair Printing Co., 1937.

The present edition of this volume has a new chapter of thirty pages with a bibliography of sixty-one references on the orthoptic treatment of strabismus. The method of Cantonnet is described at length, and there are outlines of the procedures used by Dobson, Peter, Guibor, Gifford and Cameron. Statistics are given as to the results obtained by various workers.

The remainder of the volume is similar to the earlier editions. One notes a great deal of unnecessary repetition throughout the text. Also, the treatment recommended for many of the pathologic conditions described in the sections on diseases of the retina, choroid and optic nerve is too briefly described. In the reviewer's opinion, therapy should have been discussed more fully or should not have been considered in the volume.

W. F. DUGGAN.

Nursing in Diseases of the Eye, Ear, Nose and Throat as Practiced at the Manhattan Eye, Ear and Throat Hospital, New York. Sixth edition. Price, $2.25. Pp. 288, with 93 illustrations. Philadelphia: W. B. Saunders Company, 1937.

In this edition, the sixth, the part on the eye, written by Dr. David Webster, has been revised and brought up to date. In the eighty pages devoted to the eye, the anatomic and physiologic features and the diseases of the eye are described. Then follow a discussion of the common remedies and a chapter on eversion of the lid, retractors, drops, solutions, ointments and salves. Contagious diseases of the eye, the nurse's duties at operations and instruments and appliances for the eye are the subjects of the final chapters.

A valuable part of the book is the chapters on antiseptics, sterilization and the nurse and her duties.

The text is clear, and the illustrations are well selected, so this small manual should serve as a useful guide for nurses.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6 Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of Egypt

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Time: March 1938.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31 East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.
Place: New York. Time: Oct. 6-8, 1937.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. William Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 22-25, 1938.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501—7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Place: Indiana, Pa. Time: Oct. 21, 1937.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. John King, Thomasville, Ga.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta, Ga.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406—6th Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines. Time: Nov. 18, 1937.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221—5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514—6th St., Valley City.
Place: Bismarck. Time: May 1938.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 9, 1937.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Kate Savage Zerfoss, 165—8th Ave. N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, 25 Prospect Ave., N. W., Cleveland.
Secretary: Dr. L. G. Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davisson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. J. D. Carroll, 102—3rd St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.